THESIS
FOR THE AWARD OF DEGREE OF
MASTER OF SURGERY (General Surgery)
SESSION 2020-2023

"ANATOMICAL VARIATIONS OF EXTRAHEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLECYSTECTOMY - AN OBSERVATIONAL STUDY"

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DEPARTMENT OF GENERAL SURGERY
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कामयाबी तक जाने वाले रास्ते सीधे नहीं होते ह4। लेिकन कामयाबी िमलने पर सभी रास्ते सीधे हो जाते ह4।

- आचाय चाण=

Having completed my under-graduation more than six years ago, I have had roller coaster ride of my life before I could attain my place in this institution for the degree, I like many other students, of my dreams. All my family, relatives, friends, seniors, employers and all other well-wishers, whom I have been blessed to have in large numbers, tried to convince me, either out of love or anger or frustration or reasoning, to either give up on my dream subject or compromise on it and look for alternatives.

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Dr Tanuj Khanna

(Batch 2020-2023)

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INTRODUCTION

INTRODUCTION

Extra-hepatic biliary tract (EHBT) surgery is one of the most popular surgical locations. The incidence of anatomical changes in this system has been observed to range from 7.3 percent to 47 percent.[1]

Variations in the architecture of the gallbladder, bile ducts, and the arteries that feed them, as well as the liver, are critical for the surgeon to notice since failure to do so may result in unintended ductal ligation, biliary leaks, and strictures during laparoscopic cholecystectomy.[2,3] Congenital extrahepatic biliary tree abnormalities have long been known, despite the fact that they are rare[4] and may be clinically significant[5] because they may startle surgeons during laparoscopic cholecystectomy.[6] These include anomalous or auxiliary biliary ducts, abnormal cystic ducts, bile duct cysts, biliary tract alteration associated with situs inversus, and an improper attachment of the bile duct to the pancreatic duct, as well as vascular abnormalities.[6] There are a few less frequent and more complex anatomic variants as well.[7]

Cholelithiasis is a frequent surgical condition across the globe, and cholecystectomy provides a full cure. Although cholecystectomy is one of the most frequent major surgical operations, it often surprises surgeons owing to the high prevalence of extrahepatic biliary tree congenital abnormalities.

Cholelithiasis, cholecystitis, gallbladder atresia, and malignant neoplasms of the gall bladder or otherportions of the biliary tract are the most frequent extrahepatic biliary system illnesses.[9] In humans, jaundice is typically the first symptom of illnesses affecting the gallbladder or extrahepatic biliary organs; abdominal effusion may suggest bile peritonitis. A greater bilirubin content in an effusion compared to serum (>10-fold difference) indicates bile leaking into the abdominal cavity and is a surgical urgency. [10]

Surgeons must be aware of any differences in the position and course of the region's vascular supply for regular cholecystectomy and operations requiring biliary tree exploration. The cystic artery, which is normally anterior to the CBD and superior to the cystic duct, is occasionally paired. It might be just next to the cystic duct. [11]

Variations and abnormalities in the vascular supply to the gallbladder and

extrahepatic biliary tree are also prevalent, posing considerable problems to surgeons. The superior mesenteric artery may provide an auxiliary right hepatic artery to the hepatic artery.

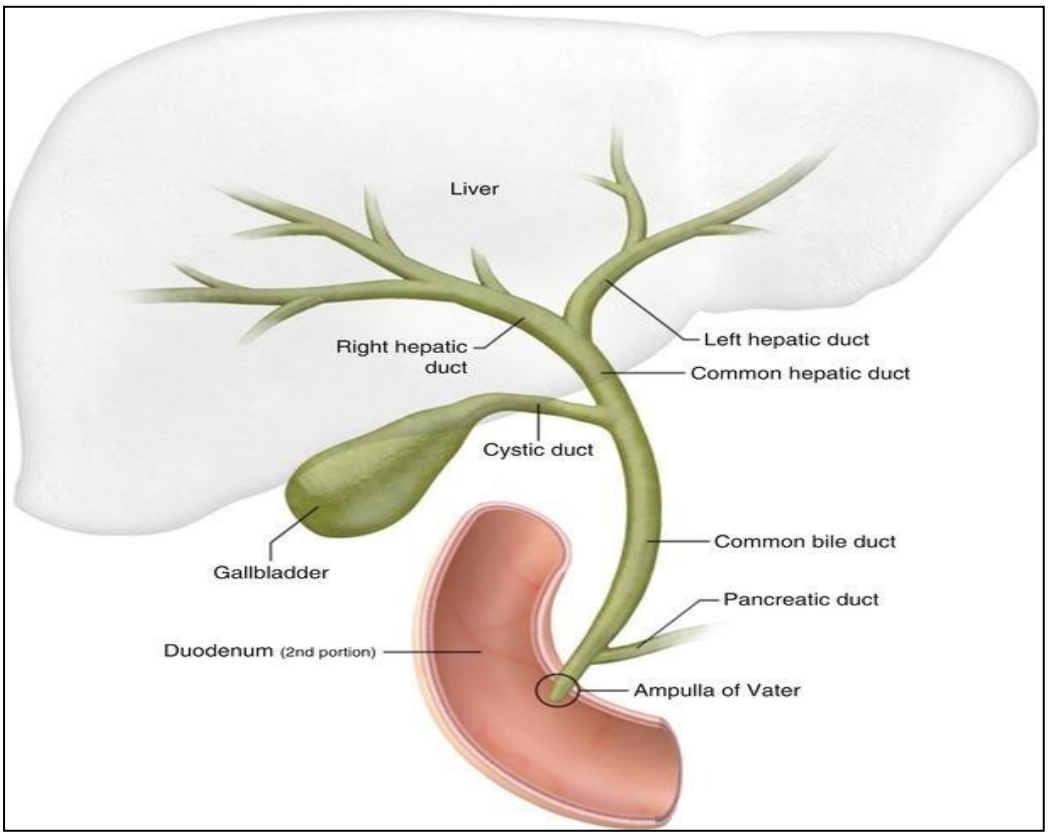


Figure 1: Anatomy of the Extra hepatic biliary tree

The hepatic artery takes an irregular path when it passes prior to the hepatic duct or when the right branch is located anterior to the right hepatic duct. Clinicians should be aware of the possibility of venous varicosities or aneurysmal dilations of the right hepatic artery in Calot's triangle.

J.F. Calot identified a triangle region in 1891 that included the cystic duct, right hepatic duct, and bottom margin of the liver. [12] In 1981, Rocko et al. reported the probable changes in the Calot's triangle area. [13] Hugh et al. proposed in 1992 that Calot's triangle be renamed the hepatobiliary triangle, with the tiny cystic artery branches feeding the cystic duct referred to as Calot's arteries. [14]The cystic artery usually starts from the right hepatic artery and runs to the right of the common hepatic duct inside the cystohepatic triangle. Bleeding of the cystic artery is a particularly difficult complication during laparoscopic cholecystectomy because it reduces overall visibility in the abdomen. The reported incidence of conversion to

open surgery due to blood vessel damage is 6.62 percent. [15]

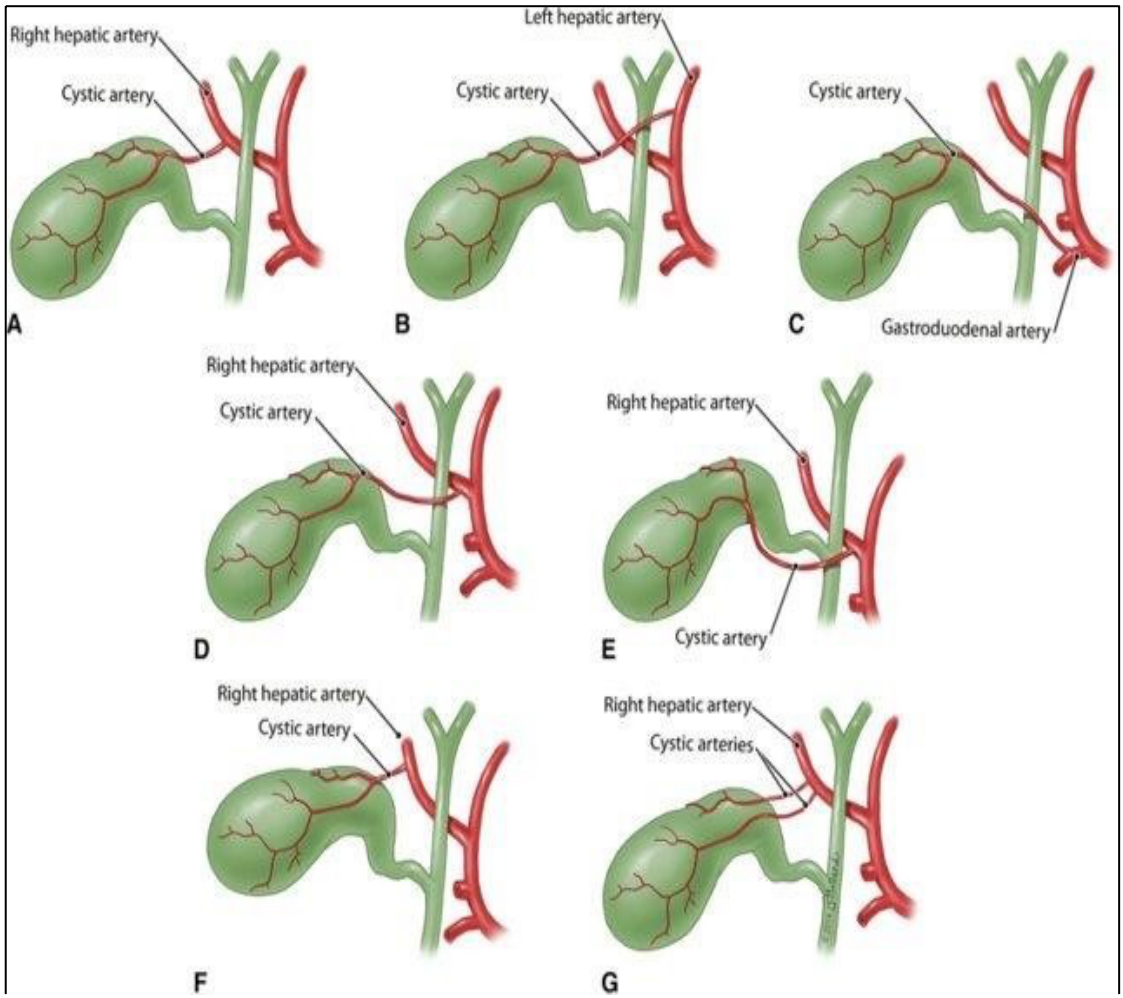


Figure 2: Most commonly found and, clinically important variations of the cystic artery[8]

Young surgeons who have just begun utilising laparoscopic techniques often struggle with reaching the Calot's triangle. Iatrogenic injuries in this area account for a significant portion of morbidity in patients undergoing open or laparoscopic cholecystectomy. As a result, it has been the primary focus of study in laparoscopic cholecystectomy for the categorization of diverse structures in Calots triangle.

An accessory cystic artery may develop from the main hepatic artery or the right hepatic artery in certain situations. Incomplete visibility before dissection of the cystic artery during open or laparoscopic cholecystectomy might result in injury to these other important arteries. The cystic artery's course and placement in relation to other structures may be very varied. The cystic artery is seen in the hepatic ducts and even behind the main hepatic duct. In

up to 95% of instances, the cystic artery starts from the right hepatic artery and travels parallel andmedial to the cystic duct.[16]

If it starts more proximally, it usually comes from the common hepatic artery (in 33% of instances) or, less often, from the celiac trunk, and in 16% of cases, the artery may lay posterior to the hepatic duct.The artery runs more parallel to the CBD in these cases, providing apparent complications for surgeons who must properly identify this vessel in preparation for and during cholecystectomy. Double cystic arteries are observed in 8-15 percent of patients, and a rare (0.3 percent) patient has a treble arterial supply to the gallbladder, while auxiliary cystic arteries are detected in 12% of patients. [17]

According to various research, these anatomical differences contribute for 9.6 percent to as much as 54 percent of the variance. As a result, we conducted this research with the goal of minimising the incidence of bile duct damage. [18] Variations in the architecture of the gallbladder, bile ducts, and the arteries that feed them, as well as the liver, are essential to the surgeon because failure to detect them may result in unintentional ductal ligation, biliary leaks, and strictures after cholecystectomy.

Prior to laparoscopic cholecystectomy, it is vital to be familiar with these variations; nevertheless, preoperative identification by normal examinations is difficult and only observed in uncommon situations, and they often turn out to be unexpected discoveries after laparoscopic surgery. Radiologic examination, on the other hand, may detect a broad range of biliary tree abnormalities as well as pancreatic anomalies. [19]

The goal of this research was to look at the structural differences in the extrahepatic biliary tree and vasculature that occur following cholecystectomy. Inadequate visibility and exposure of the cystic artery at the level of the gallbladder neck are the most prevalent surgical errors.

In most situations, surgical dissection of the cystic duct and cystic artery should commence next to or near the cystic duct's origin or around the site of entrance of the artery's branch arteries into the gallbladder wall. This is most likely the most crucial step in avoiding bile duct or hepatic artery damage during open or laparoscopic cholecystectomy. [20]

Avoiding these injuries requires proper exposure of Calot's triangle and careful identification of anatomic components. When a bile duct damage is identified, big referral hospitals with competent biliary surgeons provide the best results. Understanding the

numerous anatomic defects of the cystic duct and artery aids in directing dissection of these structures and preventing harm to the common bile duct during cholecystectomy. With advancements in medications and surgical technology, difficult surgical operations may now be performed with little morbidity and mortality. The effectiveness and safety of laparoscopic cholecystectomy, orthotopic liver transplantation, and hepatobiliary surgery are all dependent on a thorough understanding of the prevalent embryologic abnormalities of the biliary tree.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

Aim:

To study the anatomical variations of extra hepatic biliary tree and vasculature encountered during cholecystectomy.

Objectives:

- To assess the formation of common hepatic duct intra-operatively during cholecystectomy
- To assess the type of union of cystic duct with common hepatic duct intra-operatively during cholecystectomy.
- To assess the level of termination of cystic duct with common hepatic duct intra-operatively during cholecystectomy.
- To assess the length of different ducts such as cystic duct, common hepatic duct, common bileduct and any accessory duct intra-operatively during cholecystectomy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Bile, a digestive fluid generated and released by the liver, is conveyed through the biliary tree, anetwork of branching bile ducts. Each hepatocyte generates bile, which is collected by many small

tubular tubes called canaliculi. These canaliculi drain into an intralobular bile duct, which collects bile from each lobule, the liver's functional unit. Intralobular channels then drain into interlobular ducts positioned between lobules.

The interlobular ducts join to produce the liver's two major bile ducts: the right hepatic duct (RHD) and the left hepatic duct (LHD). Extrahepatically, the RHD and LHD join to create the common hepatic duct (CHD), which travels via the hepatoduodenal ligament until it meets the cystic duct, the bile duct that leads to the gallbladder. The common bile duct is formed by the union of the CHD and the cystic duct (CBD).

The hepatopancreatic ampulla, also known as the hepatopancreatic duct or the ampulla of Vater, is a spherical structure situated at the junction of the common bile duct and the pancreatic duct, indicating the entrance point of bile into the second half of the duodenum. The smooth muscle fibres of the sphincter of Oddi govern this, which opens at the duodenal papilla, enabling bile to pass into the small intestine. Alternatively, bile might enter the gallbladder and be stored there through the cystic duct.[21-23]

There are intrahepatic ducts.

- Canaliculi: Microvilli are present to improve surface area.
- Intralobular ducts: These ducts run parallel to the hepatic artery and portal vein. These three structures are known as portal triads and are surrounded by a layer of connective tissue known as Glisson's capsule.
- Ducts interlobular
- The right hepatic duct Drains the liver's right lobe (segments V, VI, VII, and VIII) Hepatic duct on the left: Drains the liver's left lobe (segments II, III, IV).
- Small ducts from both the right and left lobes drain the caudate lobe of the liver (segment I).

Ducts Extrahepatic

- Extrahepatic duct portions of the right and left hepatic ducts
- The common hepatic duct Approximately 4 cm long

- The cystic duct is the gallbladder's outflow route. The diameter is around 7 mm. Heistervalves are included.
- The common bile duct width should be less than 6 mm. It ranges from 6.0 cm to 8.0 cm. [24]

Structure and Purpose

The biliary tract's principal purpose is to carry and discharge bile. Bile is stored and concentrated in the gallbladder. Bile aids in the absorption and digestion of lipids and fat-soluble vitamins, as well as the elimination of lipid-soluble waste products. Bile is composed mostly of cholesterol, bile salts, and bilirubin. Bilirubin, which gives bile its distinctive yellow-green hue, is also expelled in bile and is responsible for faeces pigmentation.

Embryology

The biliary ducts develop from the foregut embryologically. The hepatic diverticulum emerges from the ventral foregut endodermal epithelium early in the fourth week of development. The anlage for the liver, extrahepatic ducts, gallbladder, and ventral pancreas is the hepatic diverticulum. The hepatic diverticulum is separated into two buds: cranial and caudal. The cranial bud gives rise to the intrahepatic bile ducts, whereas the caudal bud gives rise to the extrahepatic biliary tree. Notably, in order for the ducts to develop, the hepatic diverticulum must recanalize. Hepatoblasts develop into cholangiocytes, which line the intrahepatic bile ducts. [24]

Anatomy of the Extrahepatic Biliary Tract

The variability of biliary architecture is perhaps its most continuous aspect. During any biliary operation, abnormal anatomy should be anticipated and sought. The right and left hepatic ducts traditionally leave the liver and combine to create the common hepatic duct.

The left hepatic duct runs from the base of the umbilical fissure to the inferior edge of segment IV of the left lobe before connecting with the short right hepatic duct immediately below the gallbladder's infundibulum. The larger length of the left hepatic duct commonly observed offers for a more adequate target for operational biliary decompression or bypass in situations of blockage or reconstruction in the
face of cancer.

The tight association of the left and right hepatic ducts with the undersurface of the

liver hilum underscores the requirement for substantial hepatic resection observed in hilar cholangiocarcinoma. Furthermore, the small length of the right and left hepatic ducts allows a tumour in this position easy access to intrahepatic secondary biliary radicals, often blocking curative removal.

The cystic duct may vary in length and usually connects to the common hepatic duct to produce the CBD. CBD travels along the hepatoduodenal ligament, which is anterior to the portal vein and lateral to the hepatic artery. The CBD travels inferiorly, posterior to the first segment of the duodenum, thenposterior to the pancreas in a groove, which is often covered by a thin layer of pancreas.

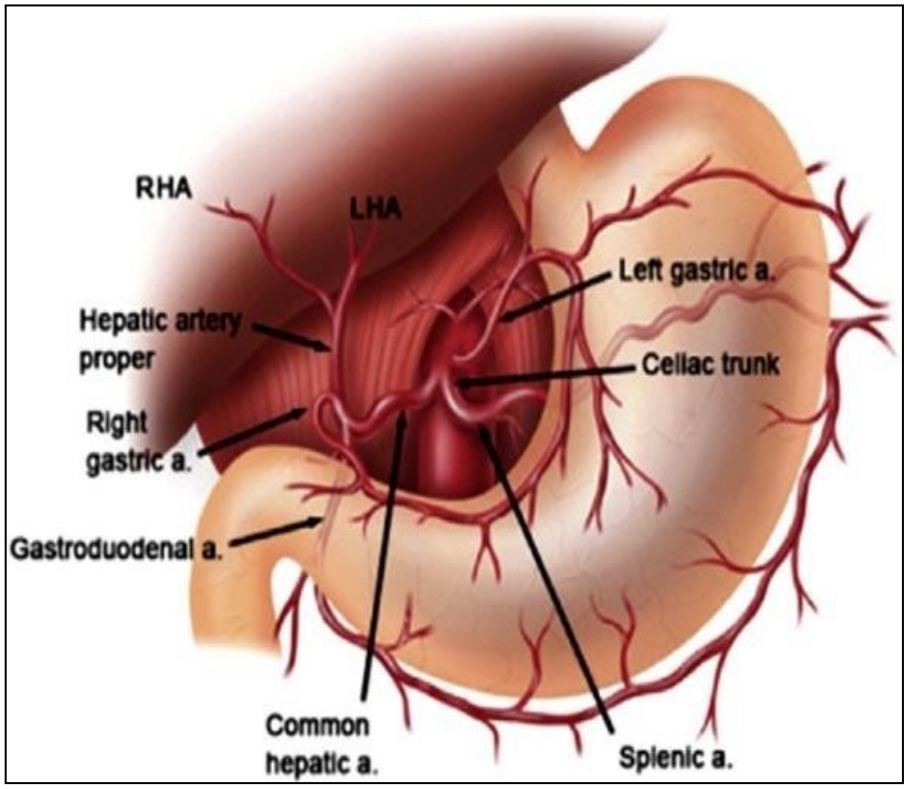


Figure 3: Classic anatomy of the celiac axis. LHA, left hepatic artery; RHA, right hepatic artery.

Finally, it reaches the duodenum's second part, either alone or after joining the pancreatic duct. [25] At normal physiologic pressure, the CBD has an interior diameter of up to 8 mm and a length ranging from 7 to 11 cm. A columnar epithelium with mucus-secreting cells lines the lumen. The CBD's primary arteries run along its lateral and medial borders and originate in the gastroduodenal and right hepatic arteries. Because disruption of this vascular network might lead to stenosis, this arterial structure is clinically significant in iatrogenic CBD damage.

The hepatic artery is closely related to the biliary tree, which is located medial to the CBD in the hepatoduodenal ligament. The celiac axis is said to branch off the aorta after it passes through the diaphragm in the traditional description. The celiac axis is divided into three branches: the left gastric, splenic, and common hepatic arteries (Fig. 3).

The gastroduodenal and right gastric arteries artery before becoming the hepatic artery proper, which ascends in the hepatoduodenal ligament, branch off the common hepatic The hepatic artery proper then divides into the right hepatic arteries before entering the liver.

The left hepatic artery travels over the medial side of the hepatoduodenal ligament before entering the left liver via the umbilical fissure. The right hepatic artery runs from medial to lateral via the hepatoduodenal ligament, usually going behind the common hepatic duct to reach the right liver.

The cystic artery, which supplies the gallbladder, is normally a branch of the right hepatic artery. A thorough awareness of the interactions between these tissues, especially aberrant architecture, is essential for general and hepatobiliary surgeons. [26]In the porta hepatis, the portal vein is located posterior to the CBD and the hepatic artery.

The superior mesenteric vein joins the splenic vein, which takes venous blood from the inferior mesenteric vein. The left gastric (coronary) vein drains the stomach's smaller curvature and empties into the portal vein near its origin. The portal vein, like the hepatic artery main, divides before entering the liver. The extrahepatic portal vein has the least variety among the portal structures, with distinct take offs of the anterior and posterior right portal branches from the main portal vein being the most prevalent aberrancy. The left portal vein before it enters the liver shows very little variation. The caudate lobe will get blood flow from the left, right, or both portal veins.

Extrahepatic Biliary Anatomy Variations

Before joining to create the common hepatic duct, the right and left hepatic ducts travel a short distance outside of the liver parenchyma. The right and left ducts seldom connect inside the liver. They may also run independently and unite lower in the hepatoduodenal ligament (Fig. 4)

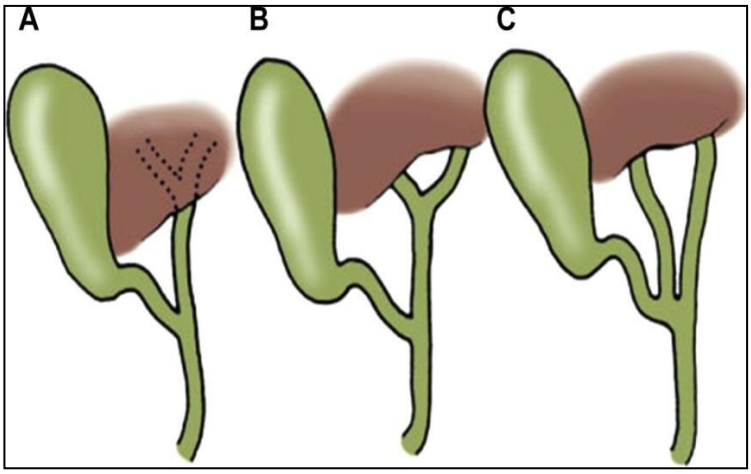


Figure 4:Variation in confluence of right and left hepatic ducts. Intrahepatic (A), extrahepatic/ typical (B), and low (C) confluence of the right and left hepatic ducts.

The first order branching of the right and left hepatic ducts inside the liver is particularly significant. According to a recent analysis based on radiographic imaging, there were unusual branching patterns of the right hepatic duct in 14% of patients and atypical branching patterns of the left hepatic duct in 8% of patients.[27] The right anterior and posterior segmental branches may appear in a variety of configurations, the most concerning of which is type A4, in which the right posterior segmental duct empties into the cystic duct.

The right anterior and posterior segmental branches may appear in a variety of configurations, the most concerning of which is type A4, in which the right posterior segmental duct empties into the cystic duct. Ligating this duct may result in cholestasis in the section it drains. Because of the great variation in the shape of these structures, intraoperative cholangiography may be quite useful in clarifying the anatomy.

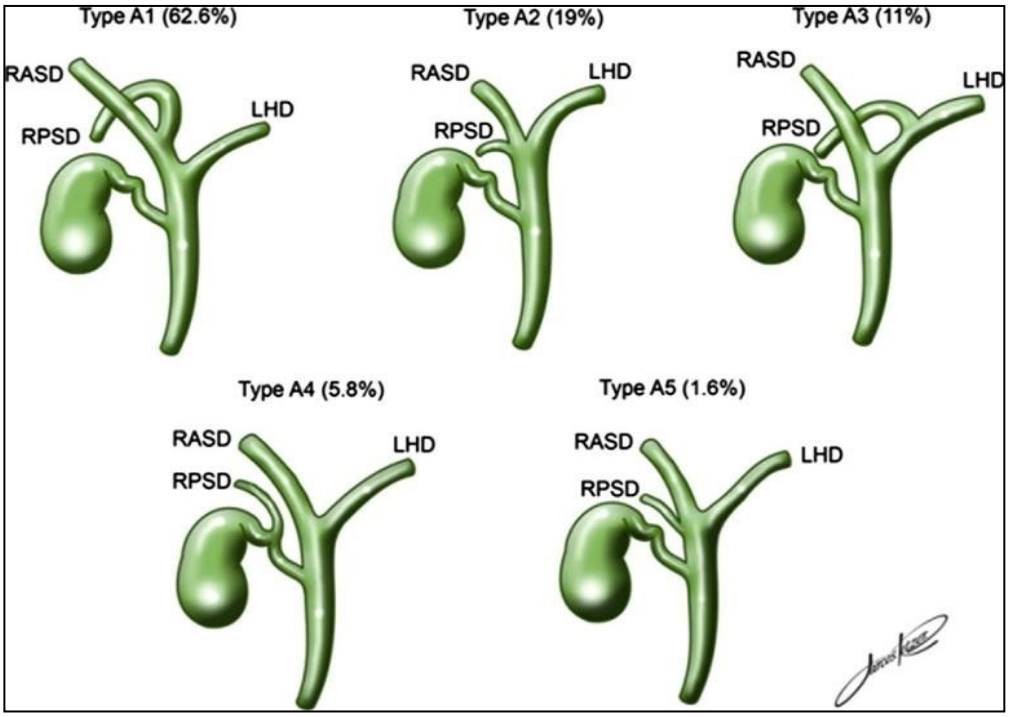


Figure 5:. Variations in sectoral drainage to right hepatic duct. LDH, left hepatic duct; RASD, right anterior hepatic duct; RPSD, right posterior hepatic duct.

Hepatic Artery Anatomy Variations

Only around 70% of individuals have traditional hepatic arterial architecture, in which the hepatic artery proper divides into the right and left hepatic arteries. Accessory right and left hepatic arteries may be present in addition to the conventional right and left hepatic arteries (Fig. 6E–G), or the right or left hepatic arteries may be replaced, indicating that they come from a different source completely.

Because auxiliary arteries normally serve a specific part of the liver, some contend that the name "accessory hepatic artery" is deceptive. Rarely, the superior mesenteric artery (SMA) entirely replaces the common hepatic artery; this is known as the totally replaced common hepatic artery (seeFig. 6A).

Before entering the liver, the right hepatic artery normally runs posterior to the hepatic duct. The right hepatic artery will be anterior to the duct in roughly one-fourth of cases (see Fig. 6H). The right hepatic artery will pass posterior to the portal vein in 10% of cases (not pictured).[28] Nearly 20% of individuals have a replacement right hepatic artery; the SMA is the most prevalent source of a replaced right hepatic artery (see Fig. 6C).

The replacement right hepatic artery may be detected ascending behind the pancreas and portal vein. The right hepatic artery gives birth to the cystic artery, which may run anteriorly or posteriorly to the hepatic duct as it approaches the gallbladder. The replacement right hepatic artery subsequently continues the normal right hepatic artery path into the liver hilum.[29]

If a replacement right hepatic artery is ligated, the surgeon must conduct a cholecystectomy since flow to the cystic artery is likely to be impeded. Ligation of the replacement right hepatic artery may potentially be harmful to a biliary enteric anastomosis because it predisposes the anastomosis to ischemia and leak. [30]

A replacement left hepatic artery will be found in roughly 15% of individuals (see Fig. 6D). The left hepatic artery will most likely begin from the left gastric artery, travel via the gastro-hepatic ligament, and reach the liver at the hilum through the ligamentum teres in these individuals. Although the replacement left hepatic artery is normally of minor importance in hepatic, biliary, and pancreatic surgery, it is important in gastric surgeries requiring gastro-hepatic ligament division. CT angiography may aid in the planning of major operations in the area by identifying vascular anomalies.

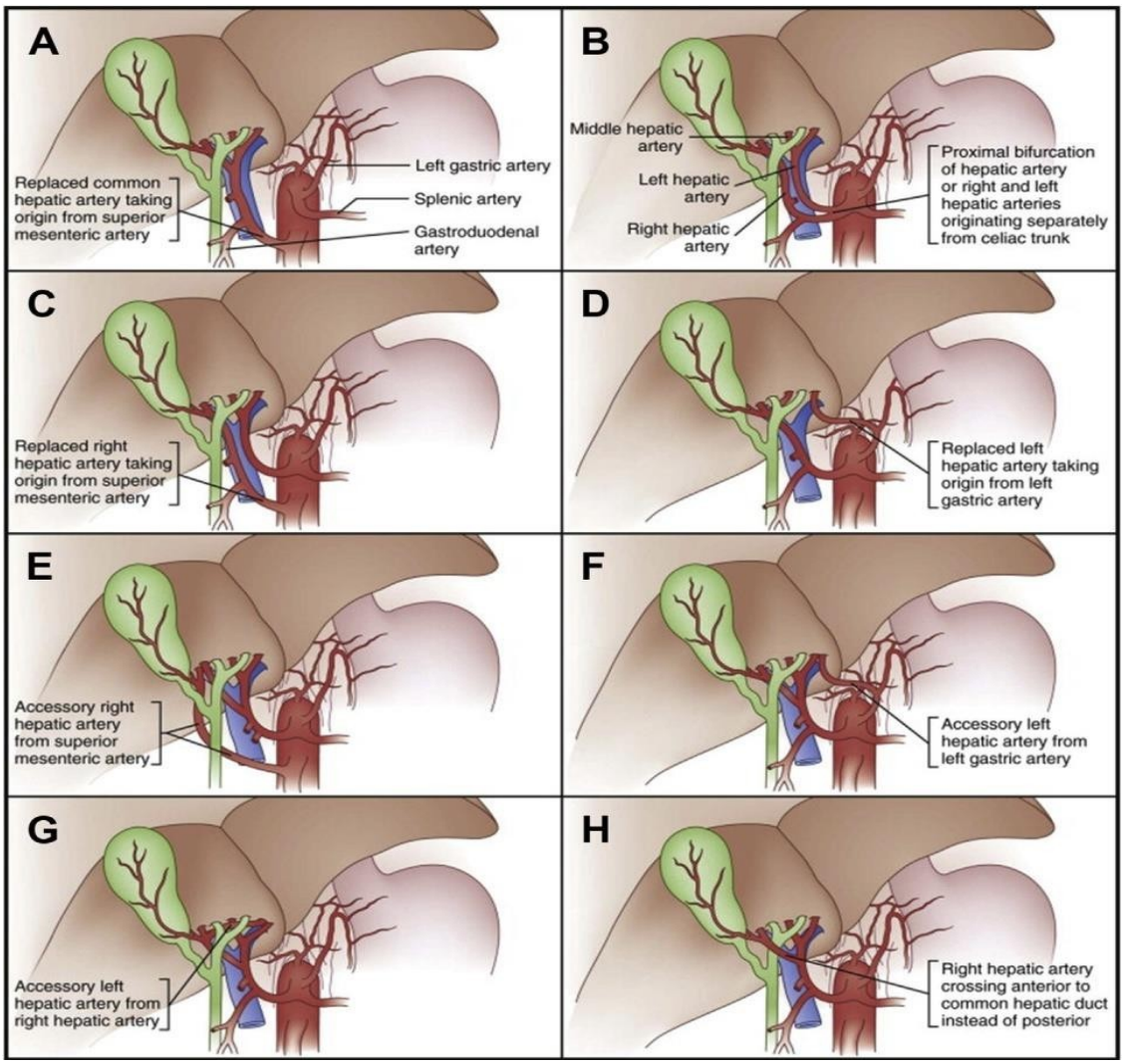

A
Replaced common hepatic artery taking origin from superior mesenteric artery
Left gastric artery
Splenic artery
Gastroduodenal artery
B
Middle hepatic artery
Left hepatic artery
Right hepatic artery
Proximal bifurcation of hepatic artery or right and left hepatic arteries originating separately from celiac trunk
C
Replaced right hepatic artery taking origin from superior mesenteric artery
D
Replaced left hepatic artery taking origin from left gastric artery
E
Accessory right hepatic artery from superior mesenteric artery
F
Accessory left hepatic artery from left gastric artery
G
Accessory left hepatic artery from right hepatic artery
H
Right hepatic artery crossing anterior to common hepatic duct instead of posterior

Figure 6: Variations in hepatic arterial anatomy

Anatomy of the Gallbladder

The gallbladder is a muscular sac that is found underneath the liver. Bile from the liver is drained to the CBD. The sphincter of Oddi's resting tone prevents bile from flowing into the duodenum and permits bile to fill the duct, with subsequent retrograde filling of the cystic duct and gallbladder. The gallbladder epithelium, which has channels that actively transport sodium chloride, concentrates the bile there. Water then follows, concentrating the bile.

The gallbladder's usual capacity is 30 mL, although it may expand to store up to 300 mL of fluid, especially in the presence of chronic distal blockage. The gallbladder wall is made up of the visceral peritoneum (on parts that do not come into direct touch with the liver), subserosa, muscularis, laminapropria, and columnar epithelium.

The fundus, body, infundibulum (Hartman pouch), and neck are the names given to the various sections of the gallbladder.[31]The cystic duct is drained via the neck. The cystic duct lumen is distinguished by mucosal folds known as Heister spiral valves. The cystic duct may drain into the right hepatic duct in a relatively short course, or it can travel alongside the common hepatic duct fora distance, entering just above the pancreas.

The lack of the gallbladder at birth has already been explored. It is important to note that the gallbladder might be intrahepatic. When investigating gallbladder agenesis or when the gallbladder isnot visible after biliary surgery, this option should be considered. Other unusual gallbladder and cystic duct abnormalities have been identified, including gallbladder duplication and left-sided gallbladder (draining into the left hepatic duct or common hepatic duct). These occurrences are quite unusual.

The cystic artery, which is generally a branch off of the right hepatic artery, supplies blood to the gallbladder. Not unexpectedly, the path of the cystic artery varies significantly. It may, on rare occasions, branch from the left hepatic artery or the hepatic artery proper, travelling anteriorly to the hepatic duct on its way to the gallbladder. It might be caused by the previously stated SMA-replaced right hepatic artery. Venous drainage of the gallbladder comprises veins that drain into the liver through the portal system as well as veins that drain directly from the gallbladder into the liver.

The gallbladder's lymphatic veins are found in the subserosal layer and drain to the Calot node and lymph nodes along the porta hepatis. Lymphatic drainage may also enter the liver directly via segments V and IVB before reaching lymph nodes in the hepatoduodenal ligament. As a result, aggressive resection for gallbladder cancer generally involves these liver parts. (fig.7)[32.]

The contraction of the gallbladder is regulated by a number of signals. Cholecystokinin (CCK) and parasympathetic innervation are the primary positive mediators of contraction. The contraction of the gallbladder is regulated by a number of signals. Cholecystokinin (CCK) and parasympathetic innervation are the primary positive mediators of contraction.

The contraction of the gallbladder is regulated by a number of signals. Cholecystokinin (CCK) and parasympathetic innervation are the primary positive

mediators of contraction. CCK is a hormonethat is released by the duodenal epithelium in response to intraluminal nutrients. CCK production causes postprandial gallbladder contraction, which transports bile to the duodenum for digestion. The vagus nerve's hepatic branch provides parasympathetic innervation, which promotes contraction.

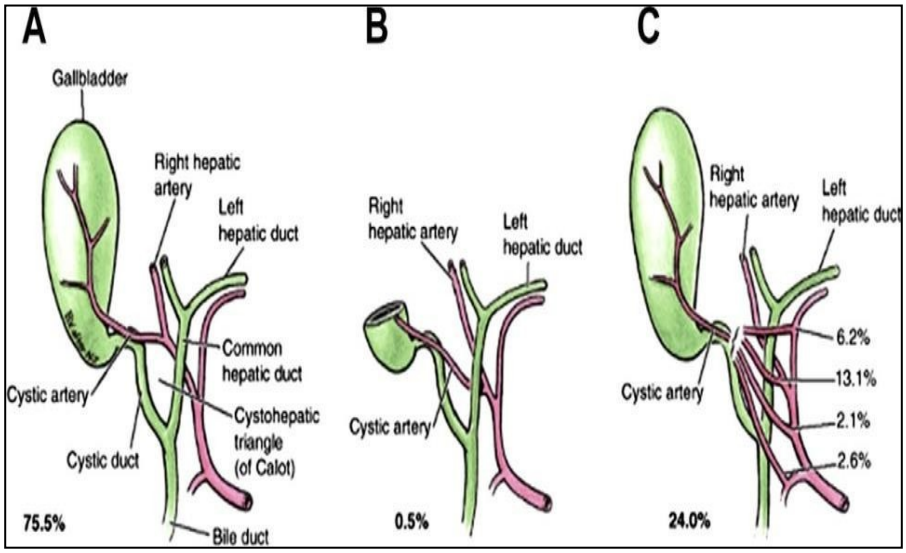


Figure 7:Variations in cystic artery anatomy.

[(*A*) Most common configuration of the cystic artery, branching from right hepatic artery. (*B*) Less common, the cystic artery is seen branching from theright hepatic artery prior to crossing posterior to the common hepatic duct. (*C*) The cystic artery is seen branch from various arteries and courses anterior to the common hepatic duct]

The gallbladder, like the rest of the digestive tract, is innervated by the enteric nervous system, which promotes cooperation with the migratory motor complex. There are other modulators of gallbladder contraction as well.

The sympathetic nervous system innervates the gallbladder via the celiac plexus. The gallbladder smooth muscle relaxes as a result of sympathetic activation. Recent research also suggests that bile components suppress gallbladder contractions through G-protein coupled receptors. Bile stasis in the gallbladder is hypothesised to contribute to the development of cholelithiasis. [33]

Considerations for Surgery

Bile duct damage is a possible consequence of cholecystectomy. The closeness of the cystic duct, the CHD and the CBD is a typical source of misidentification of key anatomical structures. The risk is larger in a laparoscopic operation than in an open procedure.[34,35] Calot's Triangle - an anatomical region defined superiorly by the inferior border of the liver,

medially by the common hepatic duct, and laterally by the cystic duct must be identified during the surgery.

The cystic artery and Mascagni's node (Lund's node) are two structures found in Calot's triangle. An auxiliary hepatic duct or right hepatic artery may also pass through this region on rare occasions. Surgeons must have a "critical perspective of safety" in order to prevent artery or biliary harm.

The critical perspective of safety consists of three components: recognising Calot's Triangle, exposing the inferior region of the gallbladder (infundibulum), and displaying just the cystic duct andcystic artery for ligation. [36]

Cholecystectomy, or gallbladder removal surgery, is frequently done laparoscopically. Cholecystectomy is indicated in cases of symptomatic cholelithiasis, cholecystitis, porcelain gallbladder, and gallbladder polyps larger than 0.5 cm. If there is an elevated risk of gallbladder cancer or other problems, cholecystectomy is advised for individuals with asymptomaticcholelithiasis. [37,38]

Clinical Importance

The biliary system transports bile into the gastrointestinal tract, which aids in the absorption of fat- soluble nutrients. In general, acute biliary blockage may produce steatorrhea, while chronic obstruction can cause fat-soluble vitamin insufficiency (A, D, E, and K). As in the case of ascending cholangitis, there is also the possibility of bacterial infection close to the site of blockage. [39]

A persistent biliary blockage, if left untreated, may lead to portal hypertension and its implications. Biliary atresia, choledochal cysts, and congenital biliary ectasia are examples of congenital biliary abnormalities. The most prevalent cause of conjugated hyperbilirubinemia in young neonates is biliary atresia. The disease's cause is unknown; however, both genetic and environmental factors have been suggested. Several causes have been hypothesised, including bile duct development failure and repetitive structural damage leading to severe luminal fibrosis.

Patients typically grow normally, but after two months of birth, they develop jaundice and Catholic stools. The first ultrasound examination is intended to rule out alternative causes of cholestasis, such as a choledochal cyst. The biliary tree's patency may be determined via hepatobiliary scintigraphy. The Kasai portoenterostomy operation, when performed early,

improves the disease's prognosis. Patients will eventually need a liver transplant. [39] Gallstones, also known as cholelithiasis, are a prevalent illness in Western societies. While the vast majority of gallstone patients areasymptomatic, biliary disease is often linked with gallstones.

A gallstone blockage of the cystic duct, for example, is the most prevalent cause of acute cholecystitis. This blockage causes gallbladder inflammation. Typical symptoms include right upper quadrant stomach discomfort, fever, nausea, vomiting, and an increased white blood cell count.

If theinflamed gallbladder irritates the subdiaphragmatic parietal peritoneum, patients may feel discomfortin the right shoulder, a condition known as "referred pain." As in Mirrizi syndrome, a blockage in the cystic duct might be so severe that the larger duct pressures on the neighbouring common hepatic duct. Mirrizi syndrome patients have jaundice in addition to the usual symptoms of cholecystitis. [40]

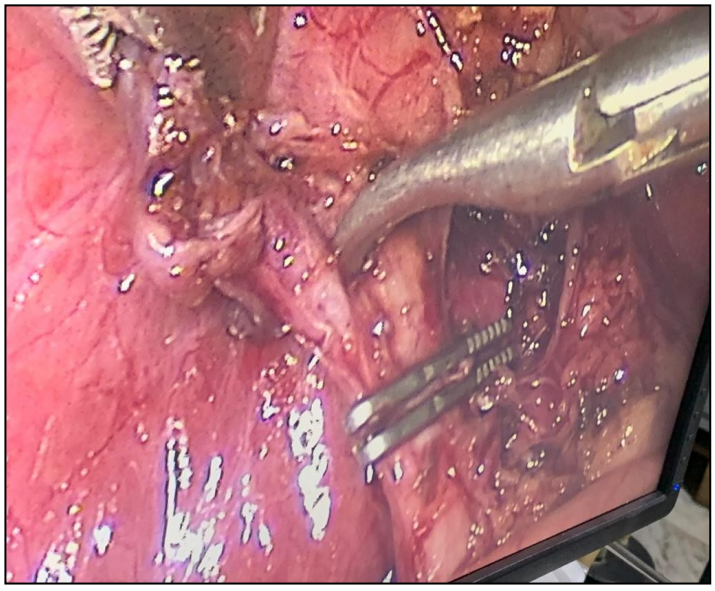


Figure 8: Double cytic duct encountered

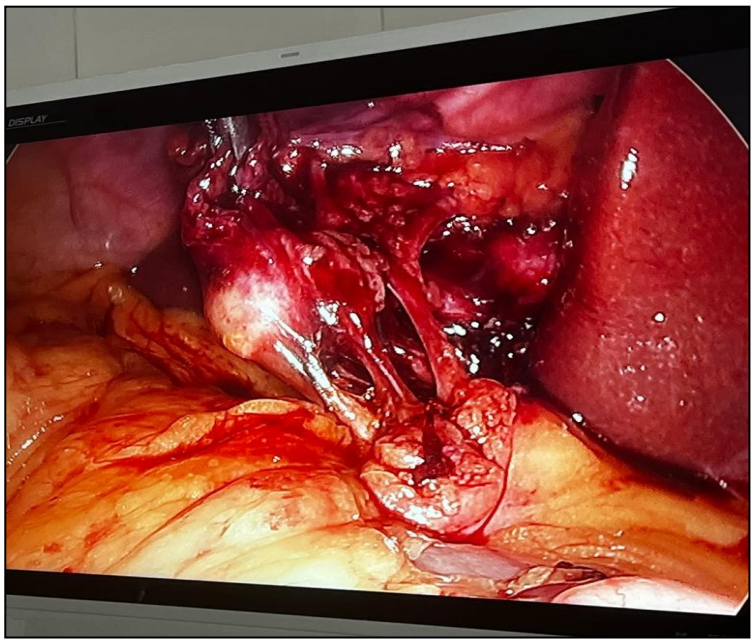


Figure 9: Both Branches of Cystic Artery Visualized.

MATERIALS AND METHODS

MATERIALS AND METHODS

PLACE OF STUDY - Department of General Surgery, Santosh Medical College andHospital

DURATION OF STUDY– July 2021 to June 2022.

STUDY DESIGN – Prospective Intraoperative observational study

SAMPLE SIZE (n)- 148 patients

$$n = \frac{Z_{\infty/2} P (1-P)}{d^2}$$

Where, $Z_{\infty} = 1.96$ at 5 % Level of significance

P = Prevalence of the anatomical variations of extrahepatic biliary tree and vasculatureencountered during cholecystectomy (10%).

d= absolute (5%)

Thereby, n =148

The study was conducted using following parameters.

I. Formation of Common Hepatic Duct

Site of union of right and left hepatic ducts

a. Extra hepatic union of right and left hepatic ducts

b. Intra hepatic union of right and left hepatic ducts

II. A. Types of union of cystic duct with common duct

a. Angular

b. Parallel

c. Spiral

B. Level of termination of cystic duct with common hepatic duct

a. High level

b. Low level

c. Normal level

III. Length of the individual ducts

a. Cystic duct

b. Common hepatic duct

c. Common bile duct

d. Any accessory duct, if any

IV. Course and arrangement of structures in Hepato-duodenal ligament

V. Variations in Ductal system

a. Presence of any accessory hepatic or cystic ducts

b. mode of termination of accessory hepatic or cystic ducts, if present

VI. Variations in arterial system related to duct system

a. Cystic artery

i. Origin

ii. Its relationship to common hepatic duct

b. Right hepatic artery

i. Origin

ii. Its relationship to common hepatic duct

VII. Calot's triangle

a. Boundaries

b. Contents

Inclusion Criteria

All patients undergoing Laparoscopic/Open cholecystectomy in general surgery department between 18-65 years of both sexes.

Exclusion Criteria

1. Patients below 18 and above 65 years of age
2. Carcinoma Gall Bladder
3. Post-surgical case of Upper Abdomen.
4. Not giving consent.

METHODOLOGY

Study Procedure

After approval from the Institutional Ethical committee all patients were selected as per inclusion and exclusion criteria. A detailed history, complete physical examination and routine & appropriate investigations were done for all patients.

Statistical Analysis

The data was entered into the Microsoft excel and the statistical analysis was performed by statistical software SPSS version 21.0. The Quantitative (Numerical variables) were present in the form of mean and SD and the Qualitative (Categorical variables) were present in the form of frequency and percentage.

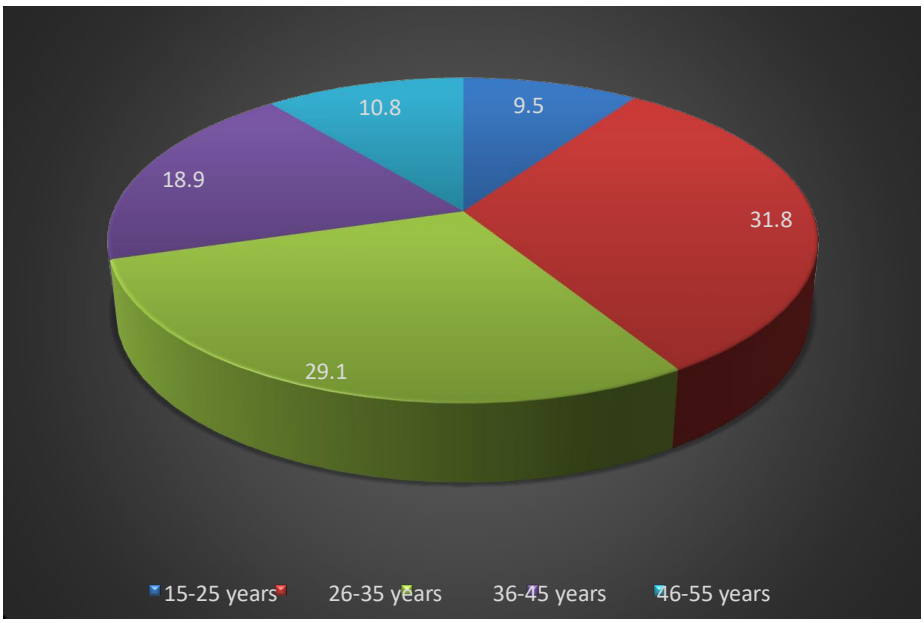
The student t-test was used for comparing the mean values between the 2 groups whereas chi-square test was applied for comparing the frequency. The p-value was considered to be significant when less than 0.05.

RESULTS AND ANALYSIS

RESULTS

Table 1: Age Distribution

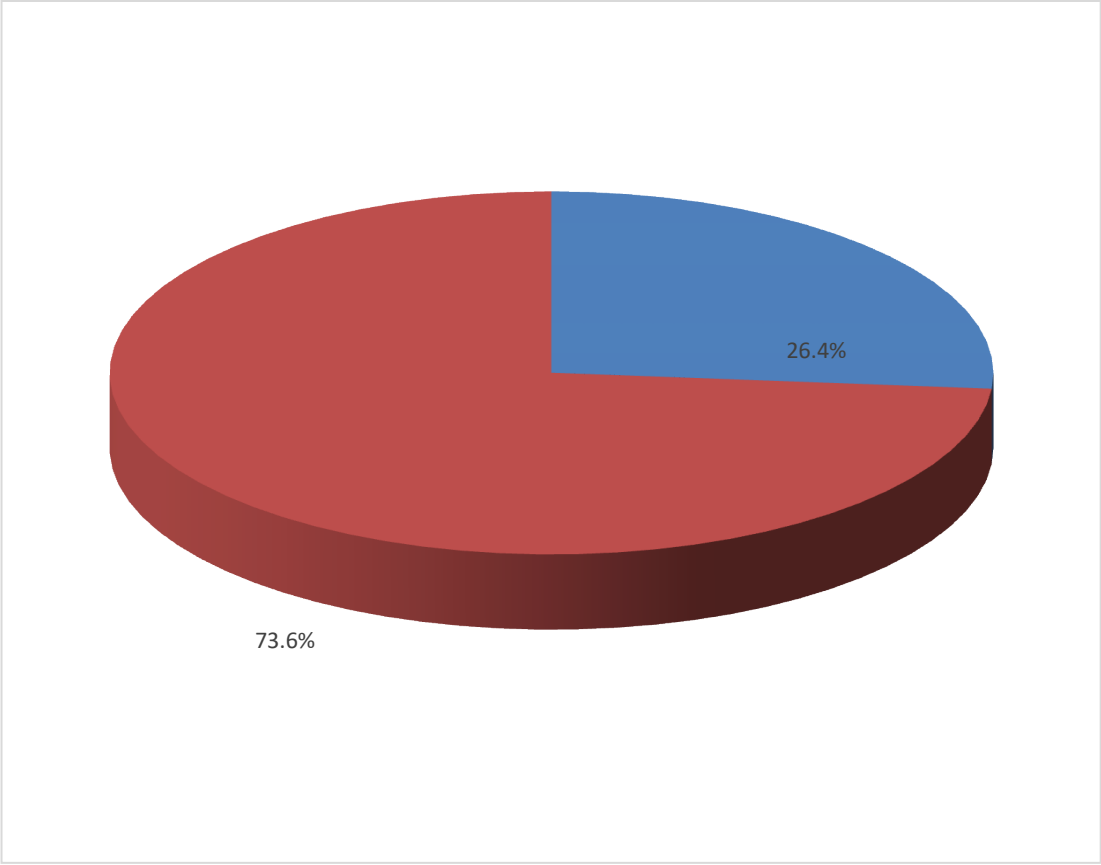
Age	Frequency	Percent
15-25 years	14	9.5%
26-35 years	47	31.8%
36-45 years	43	29.1%
46-55 years	28	18.9%
Above 55 years	16	10.8%
Total	148	100.0%
Mean±SD	40.73±12.26 (14-73)	



The age group of 148 patients was analyzed in which maximum 47 patients (31.8%) were found in age group of 26-35 years, 43 patients (29.1%) in age group of 36-45 years, 28 patients (18.9%) in age group of 46-55 years, 16 patients (10.8%) were seen above the age of 55 years and 4 patients (9.5%) were found in 15-25 years age to have gallstone disease.

Table 2: Gender distribution

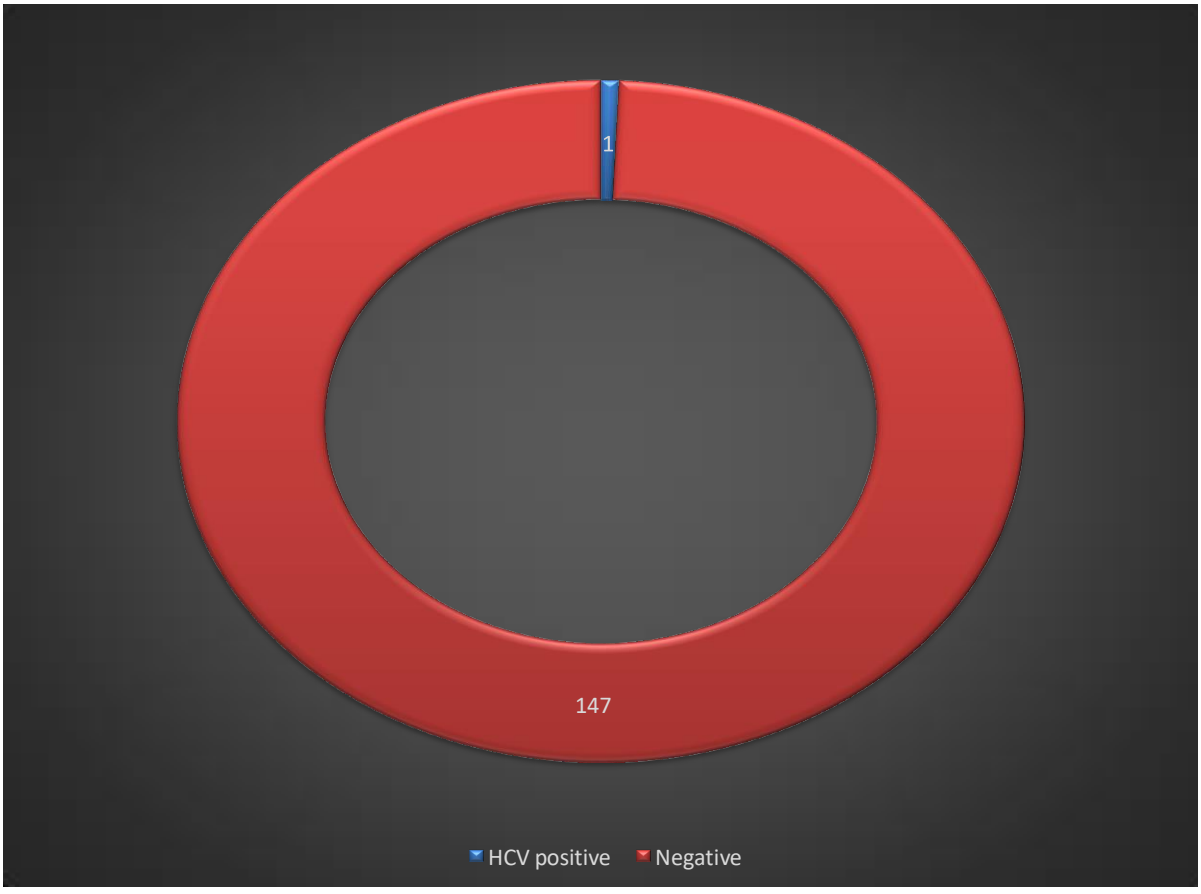
Gender	Frequency	Percent
Male	39	26.4%
Female	109	73.6%
Total	148	100.0%



It was found that there were 39 males (26.4%) patients and 109 (73.6%) females. So there was preponderance of gall stone disease in females.

Table 3: Viral markers

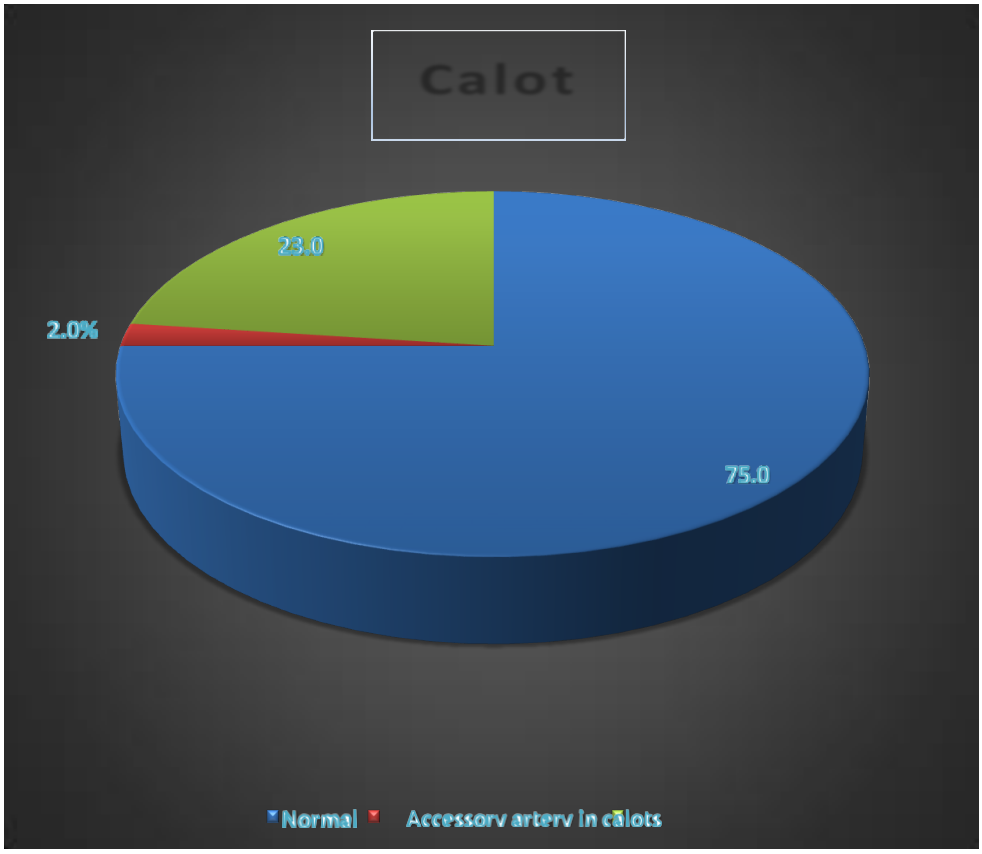
Markers	Frequency	Percent
HCV positive	1	0.7%
Negative	147	99.3%
Total	148	100.0%



Out of 148 patients one patient was found to be HCV positive (0.7%) and 147 patients where found to be HCV negative.

Table 4: Calot's anatomy (intra- operative findings)

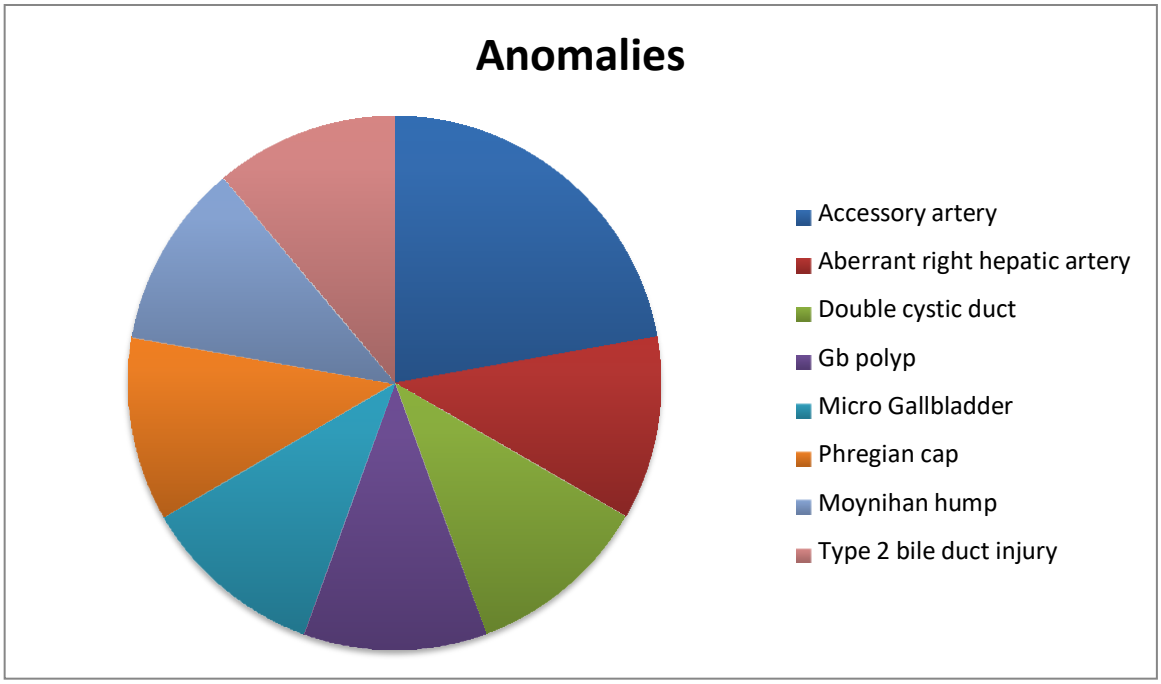
Calot	Frequency	Percent
Normal	111	75.0%
Frozen calot	34	23.0%
Accessory artery in calots	3	2.0%
Total	148	100.0%



In all the 148 patients, intra-operatively the calot's anatomy was inspected and it was found to be normal in 111 patient's (75%), frozen calot's was found in 34 patients (23%) and accessory artery in calot was found among 3 patients (2.0%).

Table 5: Anomalies

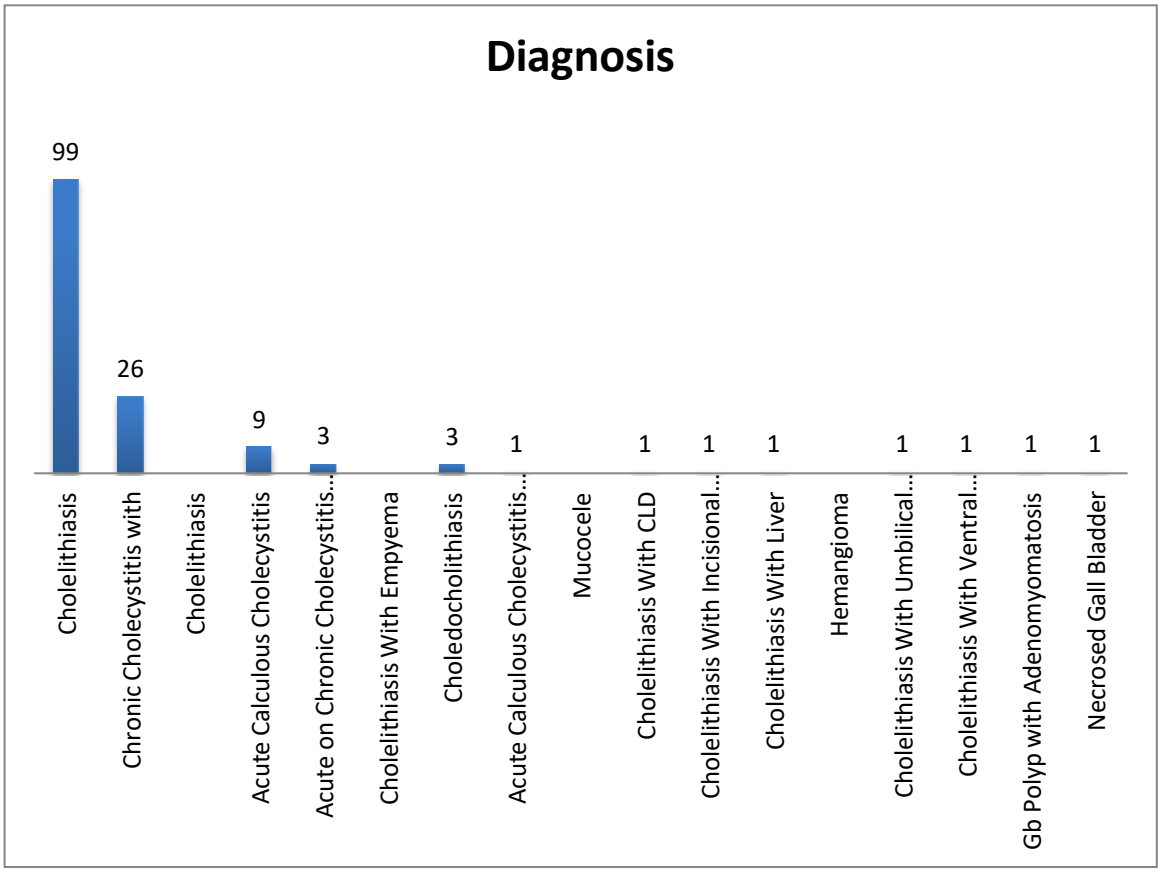
Anomalies	Frequency	Percent
No anomalies	139	93.9%
Accessory artery	2	1.4%
Aberrant right hepatic artery	1	0.7%
Double cystic duct	1	0.7%
Gb polyp	1	0.7%
Micro Gallbladder	1	0.7%
Phregian cap	1	0.7%
Moynihan hump	1	0.7%
Type 2 bile duct injury	1	0.7%
Total	148	100.0%



The Intra-operative findings during laparoscopic/open cholecystectomy of all the 148 patients were observed and noted. In 1 patient (0.7%) aberrant right hepatic artery, in 2 patients (1.4%) accessory artery, in 1 patient (0.7%) double cystic artery, in 1 patient (0.7%) gall bladder polyp, in 1 patient (0.7%) micro gall bladder, in 1 patient (0.7%) phregian cap, in 1 patient (0.7%) moynihan hump, in 1 patient (0.7%) type 2 bile duct injury was found. No anomalies where found in 139 patients (93.9%)

Table 6: Diagnosis

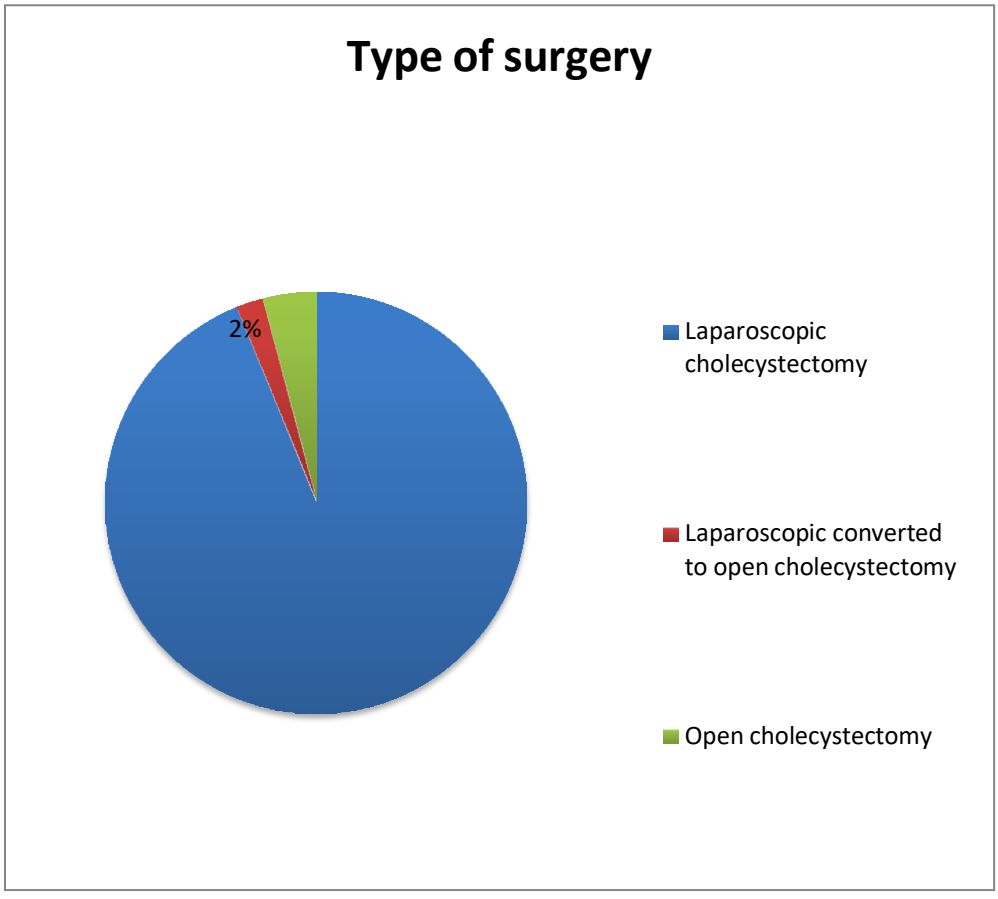
Diagnosis	Frequency	Percent
Cholelithiasis	99	66.8%
Chronic Cholecystitis with Cholelithiasis	26	17.6%
Acute Calculous Cholecystitis	9	6.08%
Acute on Chronic Cholecystitis with Cholelithiasis With Empyema	3	2.0%
Choledocholithiasis	3	2.0%
Acute Calculous Cholecystitis with Mucocele	1	0.7%
Cholelithiasis With CLD	1	0.7%
Cholelithiasis With Incisional Hernia	1	0.7%
Cholelithiasis With Liver Hemangioma	1	0.7%
Cholelithiasis With Umbilical Hernia	1	0.7%
Cholelithiasis With Ventral Hernia	1	0.7%
Gb Polyp with Adenomyomatosis	1	0.7%
Necrosed Gall Bladder	1	0.7%
Total	148	100.0%



Total 99 patients (66.8%) where diagnosed to have cholelithiasis, 26 patients (17.6%) where diagnosed to have chronic cholecystitis with cholelithiasis, 9 patients (6.08%) had acute calculus cholecystitis, 3 patients (2%) had acute on chronic cholecystitis with cholelithiasis with empyema, 3 patients (2%) had choledocolithiasis, 1 patient (0.7%) had cholelithiasis with CLD, 1 patient (0.7%) had cholelithiasis with incisional hernia, 1 patient (0.7%) had cholelithiasis with liver hemangioma, 1 patients (0.7%) had cholelithiasis with umblical hernia, 1 patient (0.7%) had gall bladder polyp with adenomyometosis, 1 patient (0.7%) had necrosed gall bladder.

Table 7: Type of surgery

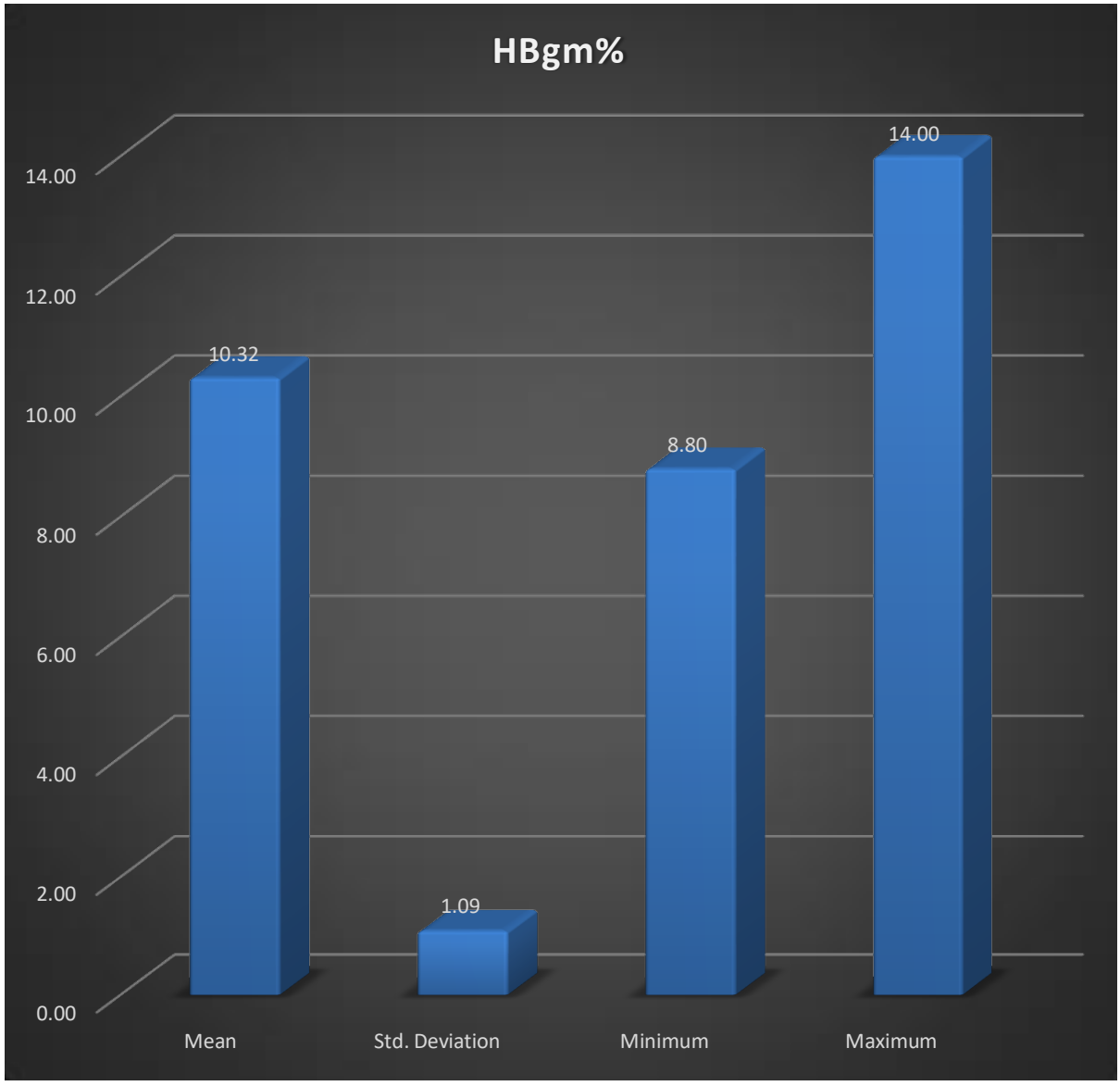
Laparoscopic/Open cholecystectomy	Frequency	Percent
Laparoscopic cholecystectomy	139	93.9%
Laparoscopic converted to open cholecystectomy	3	2.0%
Open cholecystectomy	6	4.1%
Total	148	100.0%



Laparoscopic Cholecystectomy was performed in 138 subjects (93.2%), Laparoscopic to Open Cholecystectomy in 3 subjects (2.0%), Open Cholecystectomy in 6 subjects (4.1%).

Table 8: Hemoglobin levels

	Mean	Std. Deviation	Minimum	Maximum
Hb gm%	10.32	1.09	8.80	14.00

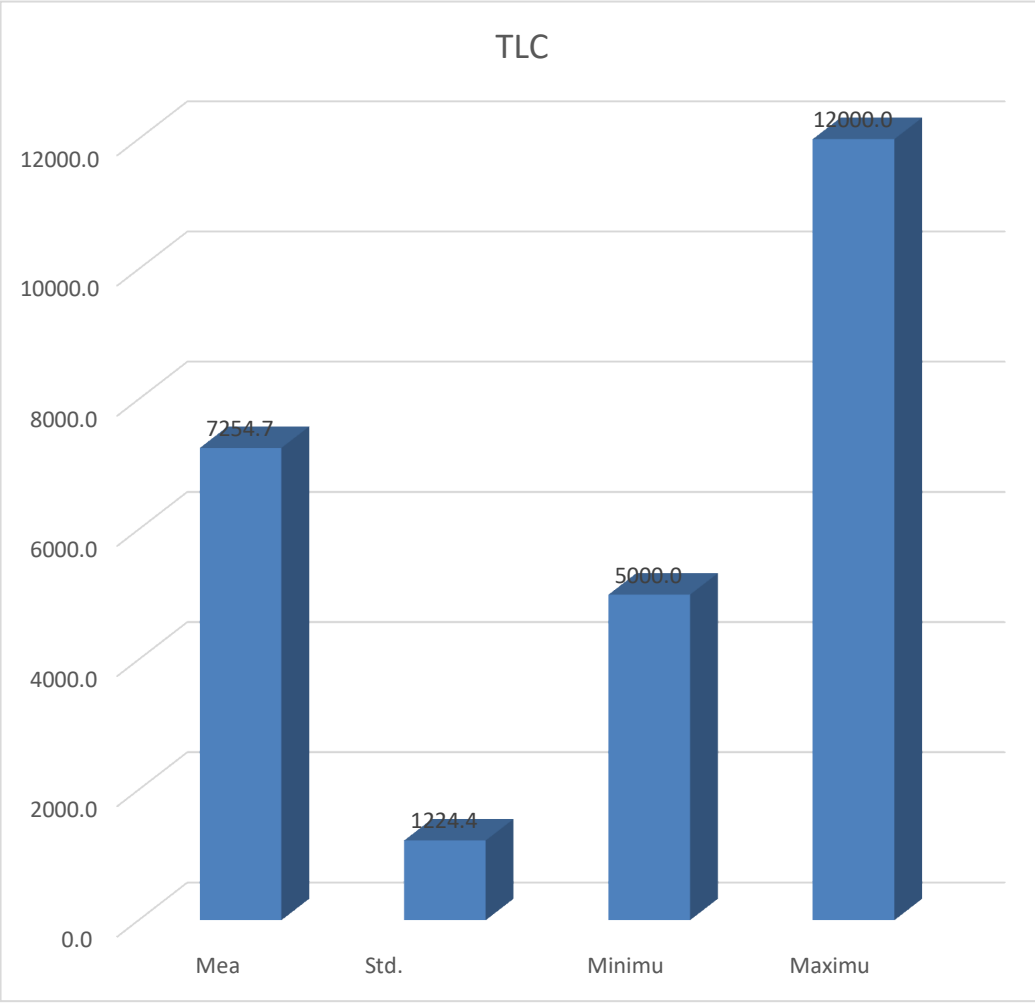


The hemoglobin levels of all the patients were analysed and, the mean (mean ± s.d.) Hb gm% was 10.32±1.09 with maximum hemoglobin level of 14.0 and minimum hemoglobin

level of 8.8.

Table 9: Total Leukocyte count

	Mean	Std. Deviation	Minimum	Maximum
TLC	7254.73	1224.49	5000.00	12000.00

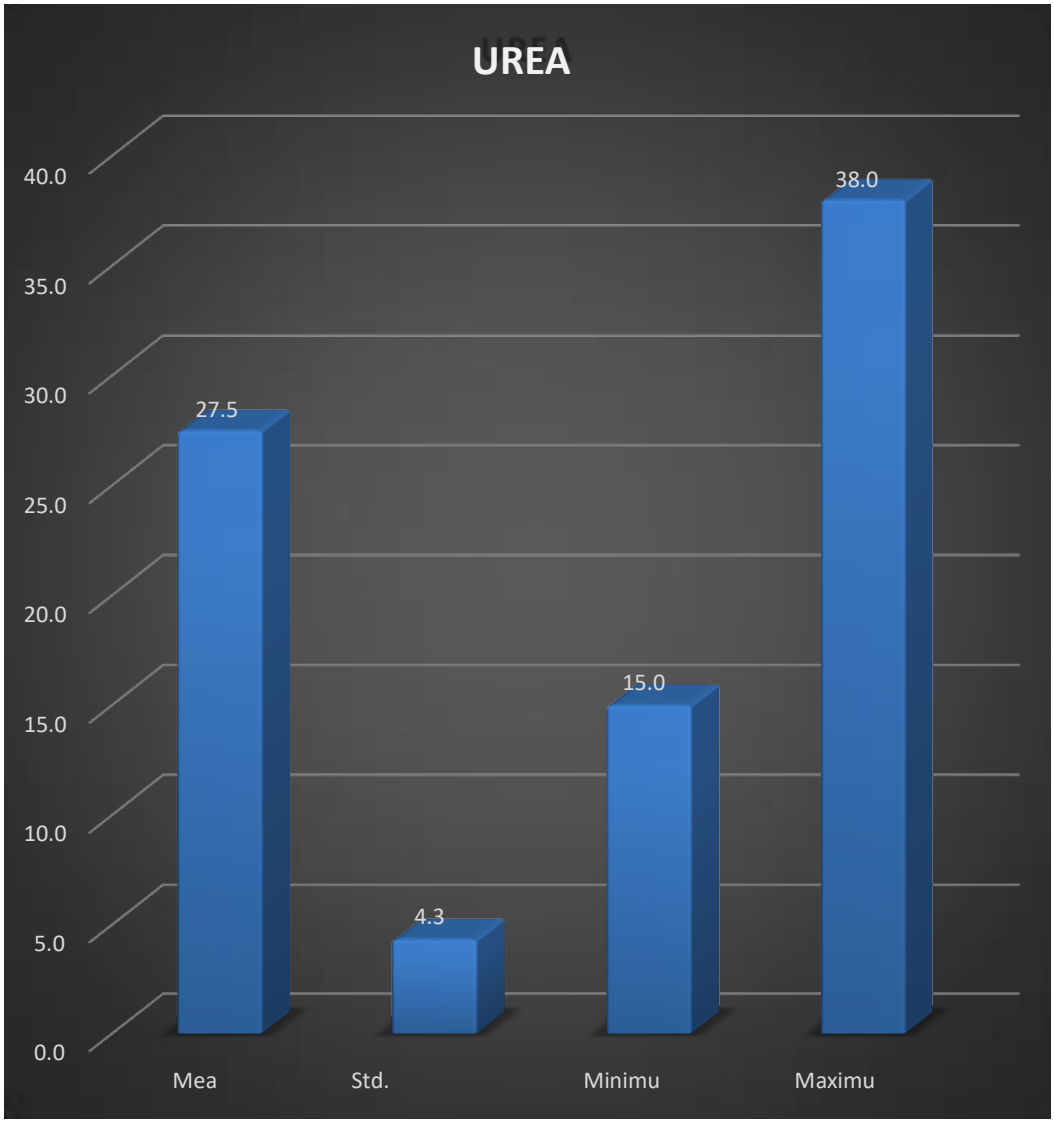


Total leukocyte count of all the patients were analysed and the mean (mean ± s.d.) TLC was 7254.73±7254.73 with maximum TLC count of 12000 and minimum TLC count of 5000

values.

Table 10: Serum Urea levels

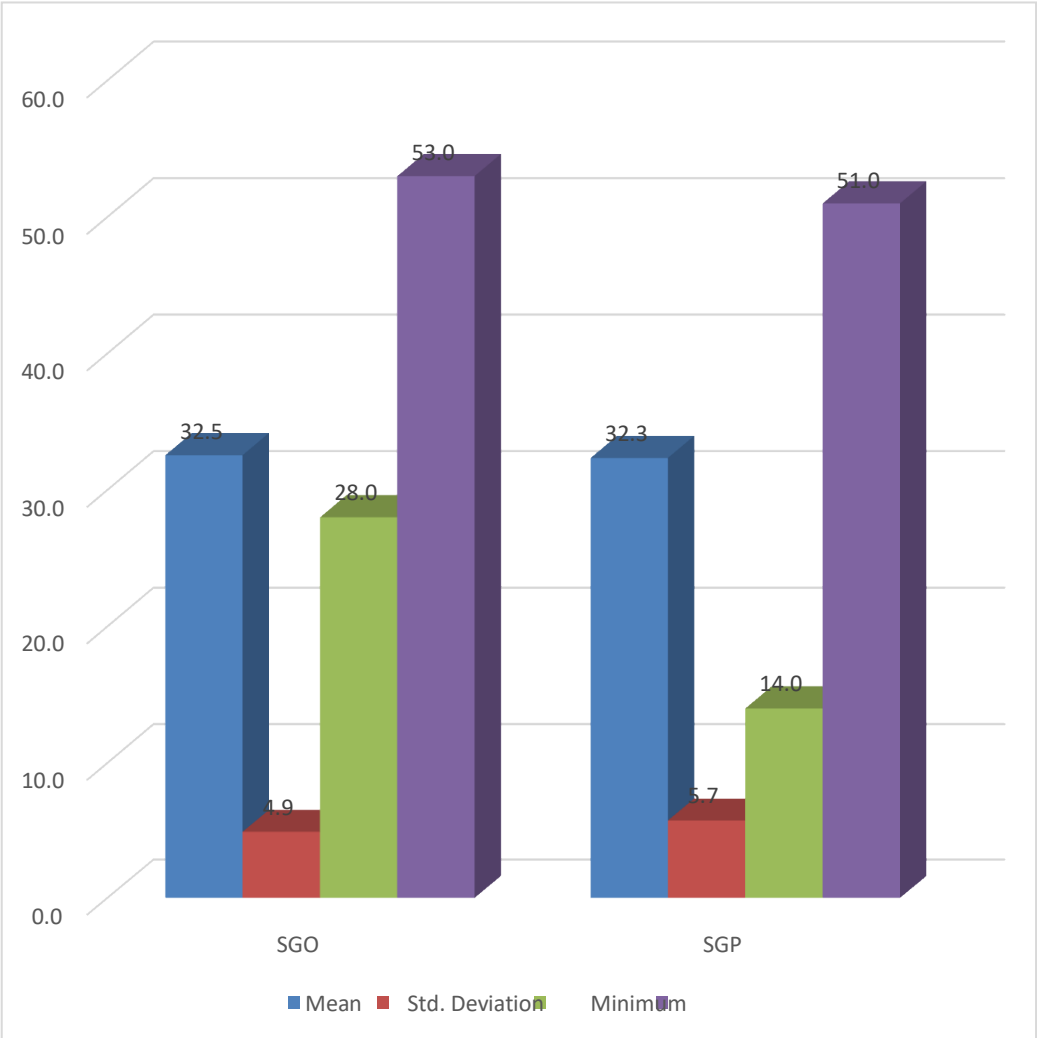
	Mean	Std. Deviation	Minimum	Maximum
Urea	27.50	4.33	15.00	38.00



Serum urea levels of all the patients were analysed, and the mean (mean ± s.d.) Urea was 27.50±4.33 with maximum serum urea level of 38 and minimum serum urea level of 15.

Table 11: SGOT, SGPT levels

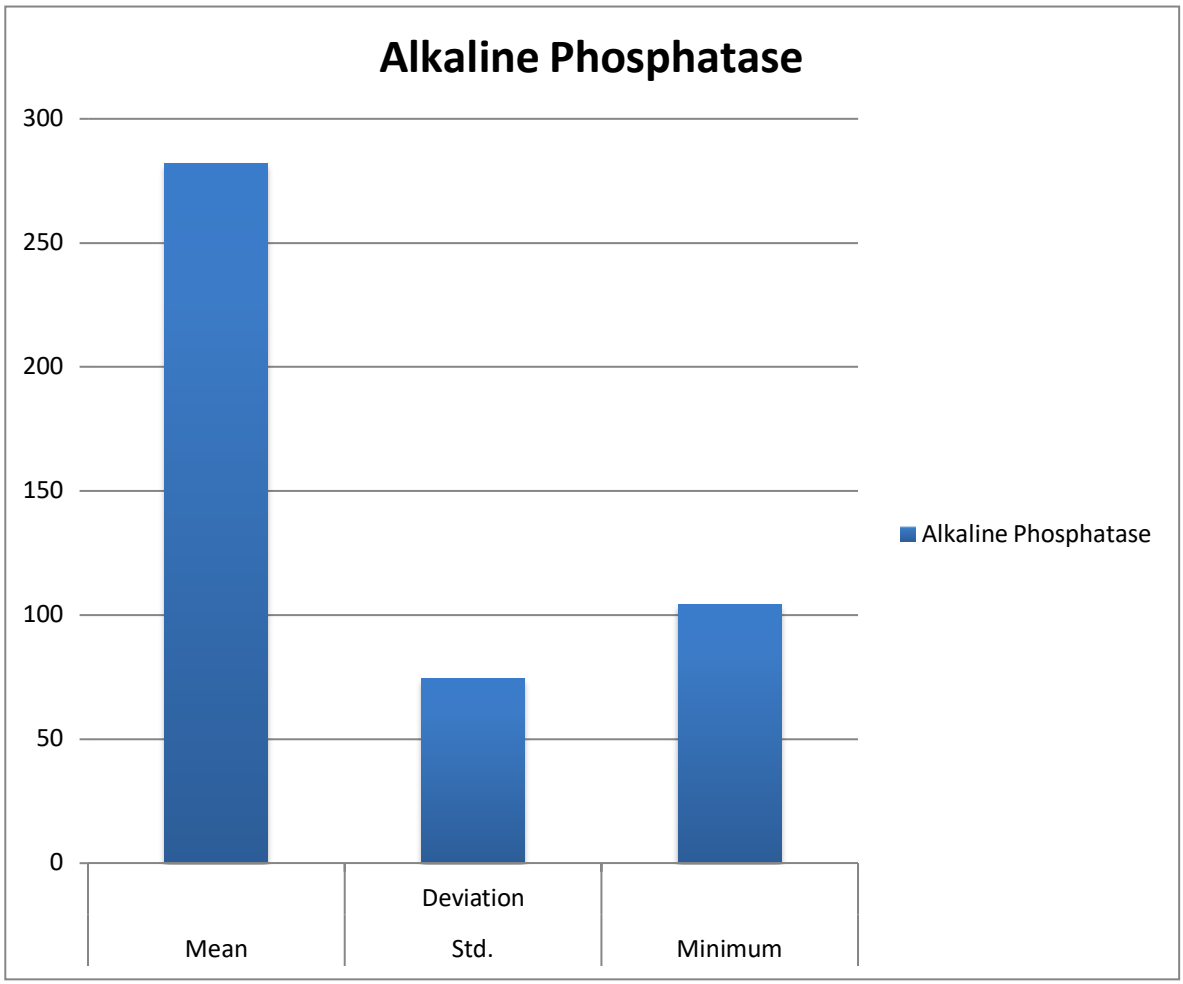
	Mean	Std. Deviation	Minimum	Maximum
SGOT	32.55	4.91	28.00	53.00
SGPT	32.35	5.75	14.00	51.00



SGOT and SGPT levels of all the patients were analysed, the mean (mean ± s.d.) SGOT level is 32.55±4.91 with maximum SGOT value of 53.0 and minimum SGOT value of 28.0 and (mean ± s.d.) SGPT level is 32.35±5.75 with maximum SGPT value of 51.0 and minimum SGPT value of 14.0 respectively.

Table 12: ALP levels

	Mean	Std. Deviation	Minimum	Maximum
Alkaline Phosphatase	281.9	74.59	104.20	186.00



ALP levels of all the patients were analysed,the mean (mean ± s.d.) ALP level is 281.9±74.59 with maximum ALP value of 186.0 and minimum ALP value of 104.2 respectively.

DISCUSSION AND CONCLUSION

DISCUSSION

The biliary tree's anatomical diversity has long been seen in surgical practice. By examining liver corrosion castings on post-mortem specimens, Couinaud initially introduced the idea of modal and aberrant anatomy in 1957. Since the addition of numerous imaging techniques (including MRCP and ERCP) to this procedure, it has become easier to visualize the architecture of the bile ducts. With the recent development in hepatic surgery's technological capabilities, it is increasingly essential for surgical success to recognize and take into account anatomic diversity in the surgical method.[67]

Variations are the result of developmental anomalies in the embryo.[68] Early in the fourth week, a ventral bud (hepatic diverticulum) emerges from the foregut's most caudal region, forming the liver, gallbladder, and biliary tree. The larger cranial part of this outgrowth, known as the pars hepatica, is the primordium of the liver, and the smaller caudal part, known as the pars cystica, expands to form the gallbladder, with its stalk becoming the cystic duct.[69,70]

This outgrowth extends into the septum transversum as rapidly proliferating cell strands, and divides into two parts as it grows between the layers of the ventral mesentery. The primordium of the gallbladder, cystic duct, and common bile duct is represented by the pars cystica, which develops in length (ductus choledochus). The caudal portion of the hepatic diverticulum lengthens, allowing the extra-hepatic biliary tree to continue developing for up to 8 weeks of gestation.

The front side of the future duodenum is where the pars cystica of the hepatic diverticulum originallystarts. The growing common bile duct's connection moves to its final location on the dorsal side of the duodenum as the duodenum rotates to the right, usually around the fifth week.

The cranial section of the hepatic diverticulum, or pars hepatica, gives rise to the hepatic duct, or ductus hepaticus. The common hepatic duct, which lacks a discernible left or right hepatic duct in the 34-day embryo, is a large, funnel-shaped organ in close proximity to the growing liver.

The dilated funnel-shaped structure at the junction of the common bile duct and cystic duct experiences a fast endodermal proliferation during the fifth week. This proliferation gives birth to multiple folds, resulting in many channels at the porta hepatis (transverse

fissure of the liver). It is hypothesized that this remodeling accounts for at least some of the many variations in the arrangement of the right and left hepatic ducts.[71] By 12 weeks of gestation, the distal sections of the right and left hepatic ducts have developed from the extrahepatic ducts and are distinct tubular structures. The earliest intra-hepatic ductal plates are the source of the proximal parts of the major hilar ducts. The layer of cells that encircles the portal vein branches like a cylindric sleeve is known as the ductal plate.[72] From the beginning of organogenesis through subsequent development, the extra-hepatic bile ducts and the growing intra-hepatic biliary tree retain luminal continuity.[71]

Age

In current study, majority (31.8%) belonged to 26-35 years followed by 36-45 years (29.1%), 46-55 years (18.9%), Above 55 years (10.8%) and 15-25 years (9.5%). *Jarrar et al*.[66] had a mean age of43 years with extremes of 11 and 83 year. *Das S. et al*.[64] found that the patients' mean age was 42.5 years, while the median age was 41.5 years.

In North-African studies, the average age of the literature ranged from 35 to 46 years.[73-75] It ranged from 16 to 89 years in other studies, with a range of 30 to 57 years.[76-78] Jarrar et al.[66] reported that age varied from 11 to 89 years old, with a mean of 49. Age and the prevalence of anatomical variations did not correlate, according to our research.

If we accept that variances are caused by anomalies in embryological development, as has previously been demonstrated, then this is totally understandable. The maljunction of the pancreatico-biliary tract, in particular, has been documented to be associated with gender-variant extra-hepatic biliary morphology.[79,80]

Gender

In present study, there were 26.4% males and 73.6% females. *Jarrar et al*.[66] stated that consisted predominantly of women (68.6%) against a male percentage of 31.4%. Sex ratio M/F was 0.46. *DasS. et al*.[64] found that 56.66 percent were girls and 43.33 percent were men, with a female to male ratio of 1.5:1. *Koshariya M. et al*.[56] stated that 72 were male and 28 were female, with 16 percent male and 10.7 percent female showing anatomical abnormalities.

According to an Italian research,[77] females were much more likely to have a variable

anatomy (45% vs. 26% in men; p=0.005). This discrepancy may be explained by the two sexes developing embryologically differently. However, a lack of evidence cannot support this idea. Jarrar et al.[66] reported that the gender ratio was 0.45, with 68.6% of the participants being women and 31.4% being men. Gender was not correlated with variant anatomy.In the study by Khan et al., 82.7% of the study's participants were female, while 17.3% were men.[81] *Das et al*.[64] stated that the female patients made up 56.66% of the patient population while male patients made up 43.33%.

Calot

In our study, Calot was frozen among 23.0% and Accessory artery in calot was found among 2.0%subjects. None of the subjects had Fever, Wound infection/ discharge and Keloid/ hypertrophic scar.

Das et al.[64] stated that Right hypochondrium pain was the most prevalent presenting symptom (73.33%), followed by right hypochondrium pain with discomfort in the epigastrium, epigastrium pain, nausea, vomiting, and dyspepsia. The common intra operative complication was bleeding which occurred in 2 patients out of 60 patients and in both the patients had anatomical variations in EHBS. Right hypochondriun pain was more prevalent in the Talapur et al.[82] study (71.67%), however the Sheikh et al study found that right hypochondriun pain was more prevalent when combined with epigastric discomfort (71.3%).

Khayat et al.[83] found that 15% of biliary tract injuries were recorded, which is greater than in earlierstudies. This might be explained by the increased prevalence of aberrant anatomy there. The authors discovered a relationship (P = 0.04) between aberrant anatomy and biliary tract damage.

Hasan et al.[51] observed the same connection with a biliary tract damage incidence of 6%. Krahenbuhl Kallman et al. both found lower incidences of 0.5% and 0.3%, respectively.[84] In order to prevent damage, it is advised that surgeons correctly identify the EHBT anatomy intraoperatively.

Anomaly

In present study, the anomalies were Aberrant right hepatic artery (0.7%), Accessory artery (1.4%), Double cystic duct (0.7%), Gb polyp (0.7%), Micro Gallbladder (0.7%), Phregian cap (0.7%), Moynihan hump(0.7%) and Type 2 bile duct injury (0.7%).

Anandhi et al.[85] stated that in 2% of subjects, it was aberrant. The main anomalies discovered were the cystic duct joining the left side of the common hepatic duct, abnormalities in the position and division of the right and appropriate hepatic artery, and abnormalities in the position and division of the cystic artery.

2% of the individuals had Calot's triangle borders that were abnormal. Accessory hepatic ducts are one of the Calot's triangle's abnormal components in 10% of patients. 2% of the participants each hada double cystic artery, an aberrant auxiliary cystic artery, and a cystic arterial branch from the right hepatic artery outside the triangle.

Shaikh B et al.[55] discovered that Intrahepatic gall bladder in 12 cases, floating gall bladder in 2 cases, phyragian cap gall bladder in 3 cases, short cystic duct in 10 cases, long cystic duct in 7 cases, accessory cholecystohepatic duct in 5 cases, Moynihans hump in 2 cases, cystic artery anterior to cystic duct in 5 cases, cystic artery posterior to cystic duct in 4 cases, short cystic 9 of the 50 patients who had these defects detected after surgery had operational issues.

Koshariya M. et al[60] reported that the most prevalent variation in extrahepatic biliary apparatus was short cystic duct, which was identified in 6% of cases, followed by intrahepatic creation of common hepatic duct by union of right hepatic duct and left hepatic duct in 3% of instances. In 1% of cases, the cystic duct was inserted into the common hepatic duct. In 1% of cases, the cystic artery originated from the left hepatic artery, and in 1% of cases, the cystic artery was anterior to the common hepatic duct. In 2% of instances, the anterior vascular arcade inthe pancreas was missing, and its origin differed in 2% of cases. In 1% of instances, the posterior pancreatic arcade was missing, and variation in origin was found in 1% of cases. Variation in pancreatic duct route was found in 22% of patients.

Sah NP et al.[59] found that anatomical variation was noted in 33 patients (9.85%). There were 5 gallbladder anomaly, 11 cystic duct anomaly, 4 right hepatic artery anomaly, and 13 cystic artery anomaly. Biliary leakage present in two cases. There was no mortality. Although congenital anomalies of extrahepatic biliary tree are not common, it can be of clinical importance and surprise ifpresent.

Devi et al.[86] discovered accessory hepatic ducts in 17% of the participants whereas Khayat et al.[83] detected them in just 3.33% of the subjects.[19,20] Since bile duct injuries occur twice as frequently during laparoscopic cholecystectomies as they do during open

cholecystectomies, it is crucial to understand the possibility of the accessory hepatic ducts and their location.

Sen S et al.[61] found that around 10% patients showed cystic duct variations, 18% showed extra- hepatic biliary tree variations, and another 7% patients showed arterial variations. They found around 10% patients (one-fourth) showed cystic duct variations, 18% showed extra-hepatic biliary tree variations, and another 7% patients showed arterial variations which is comparable and analogous to previous incidence rate.

Koshariya M. et al.[56] stated that 72 were male and 28 were female, with 16 percent male and 10.7 percent female showing anatomical abnormalities. Observed short cystic duct in 8 cases, and the second most common variation was cystic artery origin, from left hepatic artery in 3 cases and from proper hepatic artery in 1 case. Other variations included floating gall bladder in 1 case, intrahepatic union of left hepatic duct and right hepatic duct in 3 cases, low insertion of cystic duct in 3 cases, high insertion of cystic duct to common hepatic duct in 1 It was found that considerable changes in the extrahepatic biliary system and its accompanying artery supply were identified.

In 2% of cases in the *Sharma et al*.[87] investigation, an abnormal cystic artery was found. Gall bladder variants, such as intra hepatic gall bladders and short cystic ducts, are seen in 4% of individuals. In our analysis, aberrant cystic artery, which accounts for 5% of all cystic arterial alterations, was the second most prevalent anatomical variation outside of the liver.

SUMMARY

The study findings can be summarized as follows:

- Majority (31.8%) belonged to 26-35 years followed by 36-45 years (29.1%), 46-55 years(18.9%), Above 55 years (10.8%) and 15-25 years (9.5%).
- There were 26.4% males and 73.6% females.
- The mean Hb gm% was 10.32±1.09 (8.80-14.00).
- The mean TLC was 7254.73±7254.73 (5000-12000).
- The mean Urea was 27.50±4.33 (15-38).
- The mean SGOT and SGPT was 32.55±4.91 (28.0-53.0) and 32.35±5.75 (14.0-51.0) respectively.
- The mean ALP was 281.9±74.59 (104.2-186.0) respectively.
- HCV was positive among 1 (0.7%) subject.
- Calot was frozen among 34 (23.0%) subjects.
- Accessory artery in calot was found among 2 (1.4%) subjects.
- Abberrant right hepatic artery was found among 1 (0.7%) subject.
- Double cystic duct was found among 1 (0.7%) subject.
- Gb Polyp was found among 1 (0.7%) subject.
- Micro Gallbladder was found among 1 (0.7%) subject.
- Phregian cap was found among 1 (0.7%) subject and Moynihan hump was found among 1 (0.7%) subject.
- Type 2 Bile duct Injury was found among 1 (0.7%) subject.
- The predominant diagnosis was Cholelithiasis (66.9%) and Chronic Cholecystitis with Cholelithiasis (17.6%).
- The procedures performed were LAP (93.9%), LAP to Open (2.0%), Open (4.1%).
- None of the subjects had Fever, Wound infection/ discharge and Keloid/ hypertrophic scar.

CONCLUSION

Cholecystectomy revealed the majority of extra hepatic biliary system structural abnormalities. Short cystic duct and intra hepatic gall bladder changes were the most frequent anomalies, followed by cystic artery variations. Therefore, to avoid unintentional ductal cutting, ductal injuries, and bleeding issues during laparoscopic and open cholecystectomy, every surgeon should watch out for these variations. By being aware of these variations, patients will have less morbidity, conversion, and re-exploration.

BIBLIOGRAPHY

1. De Filippo M CM, Quinto S, Rastelli A, Bertellini A, Martora R, et al. Congenital anomalies and variations of the bile and pancreatic ducts: magnetic resonance cholangiopancreatography findings, epidemiology and clinical significance. Radiol Med. 2008;113(6):841-59.
2. Marcos A. Right lobe living donor liver transplantation: a review. Liver Transpl. 2000;6(1):3–20
3. Suhocki P V, Meyers WC. Injury to aberrant bile ducts during cholecystectomy: a common cause of diagnostic error and treatment delay. AJR Am J Roentgenol. 1999;172(4):955–9
4. Bayraktar Y, Balaban HY, Arslan S, Balkanci F. Agenesis of gallbladder and multiple anomalies of the biliary tree in a patient with portal thrombosis: a case report. Turk J Gastroenterol. 2006;17(3):212–5
5. Yu J, Turner MA, Fulcher AS, Halvorsen RA. Congenital anomalies and normal variants of the pancreaticobiliary tract and the pancreas in adults: part 1, Biliary tract. AJR Am J Roentgenol. 2006;187(6):1536–43
6. Carbajo MA, Martín del Omo JC, Blanco JI, Cuesta C, Martín F, Toledano M, et al. Congenital malformations of the gallbladder and cystic duct diagnosed by laparoscopy: high surgical risk. JSLS J Soc Laparoendosc Surg. 1999;3(4):319–21
7. Hirao K, Miyazaki A, Fujimoto T, Isomoto I, Hayashi K. Evaluation of Aberrant Bile Ducts Before Laparoscopic Cholecystectomy. Am J Roentgenol. 2000;175(3):713–20
8. Andall R, Matusz P, du Plessis M, Ward R, Tubbs R, Loukas M. The clinical anatomy of cystic artery variations: a review of over 9800 cases. Surg Radiol Anat. 2016;38:1–11
9. McGregor AL. A synopsis of surgical anatomy. Brownstown, MI: William and Wilkins;1963.
10. Bismuth H. Surgical anatomy and anatomical surgery of the liver. World J surgery. 1982;6(1):3-9.
11. Castaing D. Surgical anatomy of the biliary tract. HPB (Oxford). 2008;10(2):72-6.
12. Callot JF. De la cholecystecomie. Med. Frc de Paris, Dissertation, 189.
13. Rocko JM, Di Gioia JM. Calot's triangle revisited. SurgGynecolObstet. 1981;153:410–4.

14. Hugh TB, Kelly MD, Li B. Laparoscopic anatomy of the cystic artery. Am J Surg. 1992;163:593–5.
15. Kudurupaka V., Danda M.R.. Study of factors leading to conversion of laparoscopic cholecystectomy to open cholecystectomy. Indian journal of research 2019; 8(3): 1–5.
16. Northover JMA TJ. A new look at the arterial supply of the bile duct of man and its surgical implications. Br J Surg. 1979;66:379.
17. Kanno N, LeSage G, Glaser S, Alvaro D, Alpini G. Functional heterogeneity of the intrahepatic biliary epithelium. Hepatology. 2000;31(3):555-61.
18. Bergamaschi R, Ignjatovic D. More than two structures in Calot's triangle - A postmortem study. Surg Endosc. 2000;14:354–7
19. Mortelé KJ, Rocha TC, Streeter JL, Taylor AJ. Multimodality imaging of pancreatic and biliary congenital anomalies. Radiogr a Rev Publ Radiol Soc North Am Inc. 2006;26(3):715–31
20. Ress AM, Sarr MG, Nagorney DM, Farnell MB, Donohue JH, McIlrath DC. Spectrum and management of major complications of laparoscopic cholecystectomy. American J Surg. 1993;165(6):655-62.
21. Garg S, Kumar H, Sahni D, Yadav TD, Aggarwal A, Gupta T. Rare anatomic variations of the right hepatic biliary system. Surg Radiol Anat. 2019 Sep;41(9):1087-1092. [PubMed]
22. Kafle A, Adhikari B, Shrestha R, Ranjit N. Anatomic Variations of the Right Hepatic Duct: Results and Surgical Implications From a Cadaveric Study. J Nepal Health Res Counc. 2019 Apr 28;17(1):90-93. [PubMed]
23. Chehade M, Kakala B, Sinclair JL, Pang T, Al Asady R, Richardson A, Pleass H, Lam V, Johnston E, Yuen L, Hollands M. Intraoperative detection of aberrant biliary anatomy via intraoperative cholangiography during laparoscopic cholecystectomy. ANZ J Surg. 2019 Jul;89(7-8):889-894. [PubMed]
24. Vosshenrich J, Boll DT, Zech CJ. [Passive and active magnetic resonance cholangiopancreatography: Technique, indications, and typical anatomy]. Radiologe. 2019 Apr;59(4):306-314. [PubMed]
25. Kune GA. Surgical anatomy of common bile duct. Arch Surg 1964;89:995–1004.
26. Schwartz SI, Brunicardi FC. Schwartz's principles of surgery. 9th edition. New York: McGraw-Hill, Medical Pub. Division; 2010. p. 1866, xxi
27. Chaib E, Kanas AF, Galvao˜ FH, et al. Bile duct confluence: anatomic variations and its classification. Surg Radiol Anat 2013. [Epub ahead of print].

28. Agur AM, Grant JC. Grant's atlas of anatomy. 11th edition. Philadelphia: Lippin- cott Williams & Wilkins; 2005. p. 848, xv.
29. Skandalakis J, Colborn G, Weidman T, et al. Extrahepatic biliary tract and gall-bladder. In: Colborn G, Skandalakis JE, Weidman TA, et al, editors. Skandalakis' surgical anatomy. Athens (Greece): Paschalidis Medical Publications Ltd; 2004.
30. Shukla PJ, Barreto SG, Kulkarni A, et al. Vascular anomalies encountered during pancreatoduodenectomy: do they influence outcomes? Ann Surg Oncol 2010; 17(1):186–93.
31. Hall TC, Dennison AR, Garcea G. The diagnosis and management of sphincter of Oddi dysfunction: a systematic review. Langenbecks Arch Surg 2012;397(6): 889–98.
32. Craig AG, Toouli J. Sphincterotomy for biliary sphincter of Oddi dysfunction. Cochrane Database Syst Rev 2001;(3):CD001509
33. Lavoie B, Balemba OB, Godfrey C, et al. Hydrophobic bile salts inhibit gall- bladder smooth muscle function via stimulation of GPBAR1 receptors and activa- tion of KATP channels. J Physiol 2010;588(Pt 17):3295–305
34. Pucher PH, Brunt LM, Davies N, Linsk A, Munshi A, Rodriguez HA, Fingerhut A, Fanelli RD, Asbun H, Aggarwal R., SAGES Safe Cholecystectomy Task Force. Outcome trends and safety measures after 30 years of laparoscopic cholecystectomy: a systematic review and pooled data analysis. Surg Endosc. 2018 May;32(5):2175-2183. [PMC free article] [PubMed]
35. Southern Surgeons Club. A prospective analysis of 1518 laparoscopic cholecystectomies. N Engl J Med. 1991 Apr 18;324(16):1073-8. [PubMed]
36. Zarin M, Khan MA, Khan MA, Shah SAM. Critical view of safety faster and safer technique during laparoscopic cholecystectomy? Pak J Med Sci. 2018 May-Jun;34(3):574-577. [PMC free article] [PubMed]
37. Cohen JT, Charpentier KP, Beard RE. An Update on Iatrogenic Biliary Injuries: Identification, Classification, and Management. Surg Clin North Am. 2019 Apr;99(2):283-299. [PubMed]
38. Bauschke A. [Biliary system : What does the surgeon want to know from the radiologist?] Radiologe. 2019 Apr;59(4):300-305. [PubMed]
39. Coucke EM, Akbar H, Kahloon A, Lopez PP. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Sep 28, 2021. Biliary Obstruction. [PubMed]

40. Clemente G, Tringali A, De Rose AM, Panettieri E, Murazio M, Nuzzo G, Giuliante F. Mirizzi Syndrome: Diagnosis and Management of a Challenging Biliary Disease. Can J Gastroenterol Hepatol. 2018;2018:6962090. [PMC free article] [PubMed]
41. Thompson J. Arteries in the hepatic pedicle, study in statistical human anatomy. Univ Califo Pub Anat. 1933;158
42. Everette C, Macumber. HE. Anomalous and distribution of the extra hepatic biliary ducts. Ann Surg. 1942;115:472–3
43. Daseler EH, Anson BJ. The cystic artery and constituents of the hepatic pedicle; a study of 500 specimens. Surg Gynecol Obstet. 1947;85(1):47–63
44. Michels NA. The hepatic, cystic and retroduodenal arteries and their relations to the biliary ducts with samples of the entire celiacal blood supply. Ann Surg. 1951;133(4):503–24
45. Johnston E V, Anson BJ. Variations in the formation and vascular relationships of the bile ducts. Surg Gynecol Obstet. 1952;94(6):669–86
46. Larobina M.,Nottle P.D. Extrahepatic biliary anatomy at laparoscopic cholecystectomy: is aberrant anatomy important?. anz journal of surgery. 2005;75(6):392-395.
47. Khan A.H., Zaheer M.Z. Frequency of Extra Hepatic Biliary Tree Anomalies Seen During Cholecystectomy. Ann. Pak. Inst. Med. Sci. 2008;4(4): 198-200
48. Talpur KAH, Laghari AA, Yousfani SA, Malik AM, Memon AI, Khan SA. Anatomical variations and congenital anomalies of extra hepatic biliary system encountered during laparoscopic cholecystectomy. J Pak Med Assoc. 2010;60(2):89–93
49. Mariolis-Sapsakos T., Kalles V., Papatheodorou K., Goutas N., Papapanagiotou I., Flessas I. et al. Anatomic Variations of the Right Hepatic Duct: Results and Surgical Implications from a Cadaveric Study. Hindawi Publishing Corporation Anatomy Research International Volume 2012, Article ID 838179, 5 pages doi:10.1155/2012/838179.
50. Cachoeira E., Rivas A., Gabrielli C. Anatomic variations of extrahepatic bile ducts and evaluation of the length of ducts composing the cystohepatic triangle. Int. J. Morphol. 2012; 30(1):279-283.
51. Hasan MM, Reza E, Khan MR, Laila SZ, Rahman F, Mamun MH. Anatomical and congenital anomalies of extra hepatic biliary system encountered during cholecystectomy. Mymensingh Med J. 2013;22(1):20–6.

52. Paul S, Jacinth JS, Muniappan V. Variations of the Extrahepatic Biliary Tract: Cadaveric Study. IOSR J Dent Med Sci. 2013;10(1):46–50
53. Awazli LG. Anatomical Variations of Extrahepatic Biliary System. IRAQI J MED SCI. 2013;11(3):258–64
54. Anupama D, Shivaleela C, R Lakshmiprabha Subhash. a study of anatomy of extra hepatic ducts and its variations with clinical significance. Int J Anat Res 2016;4(1):2029- 2033. DOI: 10.16965/ijar.2016.138
55. Shaikh B., Baloach I.B., Shaikh S.A. Anatomical variations: Dangerous culprits behind difficult cholecystectomies. RMJ. 2016; 41(3): 316-319
56. Koshariya M., Ahirwar S.I., Khan A., Songra M.C. Study of Abnormal Anatomical Variations in Extrahepatic Biliary Apparatus and Its Related Vessels in Cadavers. Journal of Translational Medicine and Research.2016; 21(2).
57. Kostakis D., Feretis T., Stamopoulos P., Garoufalia Z., Dimitroulis D., Kykalos S., et al. A rare anatomical variation of the biliary tree Ioannis. Journal of Surgical Case Reports, 2017;5, 1–3
58. Yakura T, Hayashi S, Terayama H, Miyaki T, Nakano T, Naito M. A case of a cystic artery arising from the superior mesenteric artery with abnormal branching of the celiac trunk. BMC Res Notes. 2017;10(1):526–9.
59. Sah N, Sah S, Khaniya S, Awale L, Shah R, Agarwal C, et al. Extrahepatic Biliary Anatomy Variation Encountered During Laparoscopic Cholecystectomy. 2018
60. Koshariya M, Behram S, Singour JP, Tiwari S, Khare V. Anomalous anatomical variation in extrahepatic biliary tree and pancreas and its related vessels: a cadaveric study. Int Surg J 2019;6:3111-6.
61. Sen S, Goel S, Gaur M. Anatomical Variation of Extra-Hepatic Biliary Tree and Vasculature Encountered During Laparoscopic Cholecystectomy. Int J Contemp Surg. 2020;8(2):21–6
62. Wasi M. S. I., Fateh O., Bukhari S.M.A. Anatomical variability in the position of cystic artery and its affect on surgical approach during laparoscopic cholecystectomy. 2020.DOI: https://doi.org/10.21203/rs.3.rs-46689/v1
63. Fateh O., Wasi M.S.I., Bukhar S.A. Anaotmical variability in the position of cystic artery during laparoscopic visualization. BMC Surg (2021) 21:263 https://doi.org/10.1186/s12893-021-01270-8.

64. Das S., Nigam A., Singh R. Study of Anatomical Variations of Extra Hepatic Biliary System Found during Cholecystectomy. International Journal of Science and Research (IJSR).2021;10(2):1036-1039.
65. Muazzam M.A., Hussain S.M., Qureshi M.T.A. Anatomical variations of extra hepatic biliary system in patients undergoing laparoscopic cholecystectomy in cmh & pemh Rawalpindi. Pak Armed Forces Med J 2021; 71 (3): 916-19
66. Jarrar M.S., Masmoudi W., Barka M., Chermiti W., Zaghouani H., Youssef S. , et al. Anatomic variations of the extrahepatic biliary tree. A monocentric study and review of the literature. la tunisie medicale .2021;99 (06) : 652-661.
67. 9- Jurkovikj D. Some Anatomic-Topographic Features of Common and Right Hepatic Ducts and their Constituents. Maced J Med Sci. 2011;4(1): 44-53.
68. 10- Hayes MA, Goldenberg IS, Bishop CC. The developmental basis for bile duct anomalies. Surg Gynecol Obstet. 1958;107: 447–56.
69. 11- Lamah M, Karanjia ND, Dickson GH. Anatomical variations of the extrahepatic biliary tree: Review of the world literature. Clin Anat. 2001;14:167–72.
70. 12- Ando H. Embryology of the Biliary Tract. Dig Surg. 2010;27: 87–9.
71. 13- Roskams T, Desmet V. Embryology of Extra- and Intrahepatic Bile Ducts, the Ductal plate. Anat Rec Adv Integr Anat Evol Biol. 2008;291: 628–35.
72. 14- Van Eyken P, Sciot R, Callea F, Van der Steen K, Moerman P, Desmet VJ. The development of the intrahepatic bile ducts in man: a keratinimmunohistochemical study. Hepatol Baltim Md. 1988;8: 1586–95.
73. 15- Elhjouji A, Ali AA, Alahyane A, El Khader A, Baba H, Oueldjeddou C, et al. Variations anatomiques des voies biliaires et conséquences chirurgicales: à propos de 19 cas. J Afr Hépatol Gastroenterol. 2009;3: 137–42.
74. 16- Abdelgawad MS, Eid M. Biliary tract variants in potential right lobe living donors for liver transplantation: Evaluation with MR cholangiopancreatography (MRCP). Egypt J Radiol Nucl Med. 2012;43:53–7.
75. 17- Barsoum NR, Samie AA, Adel L, Asaad RE. Role of MRCP in assessment of biliary variants in living donor liver transplantation. Egypt J Radiol Nucl Med. 2013;44: 131–6.
76. 18- Choi JW, Kim TK, Kim KW, Kim AY, Kim PN, Ha HK, et al. Anatomic variation in intrahepatic bile ducts: an analysis of intraoperative cholangiograms in 300 consecutive donors for living donor liver transplantation. Korean J Radiol. 2003;4: 85–90.

77. 19- Cucchetti A, Peri E, Cescon M, Zanello M, Ercolani G, Zanfi C, et al. Anatomic variations of intrahepatic bile ducts in a European series and meta-analysis of the literature. J Gastrointest Surg. 2011;15: 623–30.
78. 20- Deka P, Islam M, Jindal D, Kumar N, Arora A, Negi SS. An analysis of biliary anatomy according to different classification systems. Indian J Gastroenterol. 2014;33: 23–30.
79. 21- Tashiro S, Imaizumi T, Ohkawa H, Okada A, Katoh T, Kawaharada Y, et al. Pancreaticobiliary maljunction: retrospective and nationwide survey in Japan. J Hepatobiliary Pancreat Surg. 2003;10: 345–51.
80. 22- Kamisawa T, Takuma K, Anjiki H, Egawa N, Kurata M, Honda G, et al. Pancreaticobiliary maljunction. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2009;7: S84-88.
81. Khan AS, Paracha SA, Shah Z, Tahir M, Wahab K. Anatomical variations of cystic duct encountered during open cholecystectomy. Khyber Med Univ J 2012;4:19-22.
82. Talpur KA, Laghari AA, Yousfani SA, Malik AM, Memon AI, Khan SA. Anatomical Variation & Congenital anomalies of extra hepatic biliary system encountered laproscopic cholecystectomy. J Pak Med Assoc 2010;60:89-93.
83. Khayat MF, AI-Amoodi MS, Aldaqal SM, Sibiany A. Abnormal Anatomical Variations of Extra-Hepatic Biliary Tract, and their Relation to Biliary Tract Injuries and Stones Formation. Gastroenterology Res. 2014; 7(1):12-6.
84. Krahenbuhl L, Sclabas G, Wente MN, Schafer M, Schlumpf R, Buchler MW. Incidence, risk factors, and prevention of biliary tract injuries during laparoscopic cholecystectomy in Switzerland. World J Surg. 2001;25(10):1325-1330.
85. Anandhi PG, Alagavenkatesan VN. Anatomical variations in the extra hepatic biliary system: a cross sectional study. Int J Res Med Sci 2018;6:1342-7.
86. Devi T, Krishna P. The Study of Variations of Extra Hepatic Biliary Apparatus. IOSR-JDMS. 2013;5(5):25-31.
87. Sharma V, Saraswat VA, Baijal SS, Choudhuri G. Anatomic variations in intrahepatic bile ducts in a north Indian population. J Gastroenterol Hepatol. 2008 Jul;23(7 Pt 2):e58-62.

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PROFORMA

Serial No.: Date:

Name: Age/Sex:

IPD no.: Occupation:

Address:

Final Diagnosis:

Treatment Plan: Date of surgery:

HISTORY:

Chief complaints:

History of presenting illness

Past history:

Family history:

Co morbidities:

EXAMINATION:

General physical examination

Pallor: Icterus: Cyanosis: Clubbing: Lymphadenopathy:

Edema:

Systemic examination

- CVS:
- RS:
- P/A:
- CNS:

Local Examination:

INVESTIGATIONS:

Hemoglobin(gm%): Blood sugar(mg/dl): RBS:

Viral Markers: FBS:
PPBS:

LFT:

Serum Urea(mg/dl): Serum Creatinine(mg/dl):

OPERATION DETAILS:

Date and Time

Procedure

Anesthesia

Intra op Findings:

- Formation of common hepatic duct
- Type of union of cystic duct with common hepatic duct
- Level of termination of cystic duct with common hepatic duct
- Length of ducts:
    - Cystic duct
    - Common hepatic duct
    - Common bile duct
    - Any accessory duct
- Variations in arterial system
- Calot's triangle

SANTOSH MEDICAL COLLEGE AND HOSPITAL (GHAZIABAD)

INFORMED CONSENT

I_____Son/Daughter/Wife of_____resident of_____ hereby declare that I give my full, free and voluntary informed consent in the thesis - "**ANATOMICAL VARIATIONS OF EXTRAHEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLECYSTECTOMY".** Dr._____ has informed me to my fullsatisfaction, in the language I understand, about the purpose, nature of study and various laboratory investigations for the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.

I give full consent for being enrolled in the above study and I reserve my rights to withdraw from this study, whenever I wish without prejudice of my right to undergo further treatment at Santosh Hospital, Ghaziabad U.P.

Patient's/ Patient's relative's signature Or thumb impression

Doctors signature

Name:

Date:

We have witnessed that the patient signed the above form in the presence of his free will afterfully understanding its content.

Signature of Witness

Name

Signature of Investigator
Name: Dr. Tanuj Khanna
Designation: PG Department of General Surgery
Contact No.- 9818245096

संतोष मेडिकल कॉलेज एवं अ2ताल (गाजियाबाद)

सहमितपा (सूचित)

म4_____बेटा/ बेटी/ पBी_____

िनवासी _____ घोषणा करता Eं िक डॉ ._____, ने मुझे मेरी भाषा मL अपने अNयन "ANATOMICAL VARIATIONS OFEXTRAHEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLECYSTECTOMY" केउQे R केबारे मL, और अNयन केदौरान अपनाई जाने वाली िविभW Xयोगशाला जांच के बारे मL सूचित कर िदया है । मुझे अNयन की अविध और उसके दौरान अपनाए जाने वाले िनद^श भी मेरी भाषा मे समझा िदए गए ह4 | म4 उपरो` अNयन ML िबना िकसी दबाव केहाaखला ले रहा Eं और पूण; सहमित दे रहा Eं | सcोष अdताल, गाजियाबाद मL अपने आगे केउपचार पर िबना Xितकू ल Xभाव डाले इस अNयन से और Xयोगशाला जांच की Xकृ ित से म4 जब चाEँबाहर जाने के अपने अिधकार को सुरिhत रख रहा Eं |

रोगी /रोगी के irjेदार के ह.Ihर डॉkर के ह.Ihरनाम
िदनांक:

मेरी उपamथित मL मरीज ने पूरी तरह से डाकटर केबताए गए अNयन और िनद^ शों को समझने केबादअपनी otंp इqT से ऊपर के फाम पर ह.Ihर िकए ह4।

ह.Ihर

नाम: डॉ. तनुजखWा
पदनाम: पीजी

ETHICAL CLEARANCE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[45] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"ANATOMICAL VARIATIONS IN EXTRA HEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLECYSTECTOMY",** submitted by **Dr. Tanuj Khanna,** MD/MS student of Batch 2020-2021, Department of **General Surgery**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"ANATOMICAL VARIATIONS IN EXTRA HEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLECYSTECTOMY", Dr. Tanuj Khanna** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR, DELHI

Dr. Tanuj Khanna
MD/MS student of batch 2020-2021
Department of General Surgery
Email: drtanujkhanna9@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. Shalabh Gupta (Guide)
7. HOD of **General Surgery**

PLIAGARISM CERTIFICATE

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BIOSTATISTICS CERTIFICATE

TO WHOM SO EVER IT MAY CONCERN

CERTIFICATE

This is to certify that the results of the research work titled **"ANATOMICAL VARIATIONS OF EXTRAHEPATIC BILIARY TREE AND VASCULATURE ENCOUNTERED DURING CHOLYCESTECTOMY"** carried out by **Dr. Tanuj Khanna** from Department of Surgery, Santosh Medical College & Hospital, Ghaziabad, U.P. was analyzed statistically using software SPSS version 25.0 (IBM SPSS Statistics, IBM corp., 2018).

The necessary statistical tests were applied as per the requirement of the thesis and to the best of my knowledge.

Dr. SUKHVINDER SINGH OBEROI
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Dr. Sukhvinder Singh Oberoi
BDS, MDS, Cert. Biostatistics
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MASTERCHART

NO.	NAME	AGE	SEX	MARKERS	CALOTS	ANOMALY	DIAGNOSIS	HBgm%	TLC	SGOT/SGPT	UREA	CREAT	LAP/OPEN	FEVER	WOUND IN	KELOI
13003368	MRS SUDHESH	45	F	NEGATIVE	FROZEN CALOT	DOUBLE CYSTIC DU	GB POLYP WITH ADHEI	11	9000	30,35	18	NORMAL	LAP	NIL	NIL	NIL
13003371	BABY PATHAK	38	F	NEGATIVE	FROZEN CALOT	PHREGIAN CAP	CHRONIC CHOLECYSTI	10	10000	53,15	15	NORMAL	LAP	NIL	NIL	NIL
13005340	MRS SHANKUNTALA	45	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	9	6000	53,15	18	NORMAL	LAP	NIL	NIL	NIL
17090097	MR CHANDRABHAN	35	M	NEGATIVE	FROZEN CALOT	NIL	ACUTE CALCULOUS CO	11	7000	53,14	15	NORMAL	LAP	NIL	NIL	NIL
17088221	MRS MEENAKSHI	32	F	NEGATIVE	NORMAL	ABBERANT RIGHT H	ACUTE CALCULOUS CO	9	6800	50,16	18	NORMAL	LAP	NIL	NIL	NIL
17091066	MRS VIMMI	30	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11.8	7000	29,22	20	NORMAL	LAP	NIL	NIL	NIL
13005200	MR MANVEER	45	M	NEGATIVE	FROZEN CALOT	MUCOCELE	CHRONIC CHOLECYSTI	11	9000	30,22	22	NORMAL	LAP TO OPE	NIL	NIL	NIL
17091076	MRS POOJA	50	F	NEGATIVE	NORMAL	EMPYEMA	ACUTE CHOLECYSTITIS	11	12000	30,22	22	NORMAL	LAP	NIL	NIL	NIL
17157316	MRS RAMJANA	42	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,23	23	NORMAL	LAP	NIL	NIL	NIL
17152620	MR AMIT	33	M	NEGATIVE	FROZEN CALOT	NIL	CHRONIC CHOLECYSTI	10	6000	40,42	30	NORMAL	LAP	NIL	NIL	NIL
17098296	MRS SAMINA	55	F	NEGATIVE	FROZEN CALOT	EMPYEMA	ACUTE ON CRONIC CHC	11	12000	29,22	22	NORMAL	LAP	NIL	NIL	NIL
15003402	MRS GUDIA	31	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	6000	30,40	28	NORMAL	LAP	NIL	NIL	NIL
17098386	MR SANJAY	32	M	NEGATIVE	NORMAL	MUCOCELE	ACUTE CALCULOUS CH	10	9000	35,30	28	NORMAL	LAP TO OPE	NIL	NIL	NIL
13005672	MRS JYOTI	26	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	11	6000	35,38	20	NORMAL	LAP	NIL	NIL	NIL
13006672	MRS SUNITA	32	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	7000	40,42	20	NORMAL	LAP	NIL	NIL	NIL
17100103	MR GORAKH PRASAD	65	M	NEGATIVE	FROZEN CALOT	NIL	ACUTE CALCULOUS CH	10	8000	35,38	20	NORMAL	LAP	NIL	NIL	NIL
17100113	MRS NIRMALA DEVI	55	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	6600	40,41	20	NORMAL	LAP	NIL	NIL	NIL
17106181	MRS ALKA VERMA	30	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	6500	38,39	25	NORMAL	LAP	NIL	NIL	NIL
17106173	MRS ANAMIKA	28	F	NEGATIVE	FROZEN CALOT	NIL	ACUTE CHOLECYSTITIS	10	7000	40,42	20	NORMAL	LAP	NIL	NIL	NIL
17106185	MRS SONI	30	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	6000	38,39	20	NORMAL	LAP	NIL	NIL	NIL
17106584	Mr SANDEEP	32	M	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	9	12000	32,38	19	NORMAL	LAP	NIL	NIL	NIL
13009069	MR IMRAN	34	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	8000	32,38	19	NORMAL	LAP	NIL	NIL	NIL
17117528	MRS RITU	26	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	9	5000	30,37	19	NORMAL	LAP	NIL	NIL	NIL
17111850	MR DEEPAK RAI	47	M	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	11	8000	32,36	20	NORMAL	LAP	NIL	NIL	NIL
17119626	MRS RAJESH DEVI	49	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	7000	30,32	20	NORMAL	LAP	NIL	NIL	NIL
17119627	MRS SUSHI	25	F	NEGATIVE	NORMAL	NIL	ACUTE ON CRONIC CHC	9	6000	32,36	19	NORMAL	LAP	NIL	NIL	NIL
13010595	MRS ALKA	54	F	NEGATIVE	NORMAL	NECROSED GALL BL	CHRONIC CHOLECYSTI	10	7000	40,45	20	NORMAL	OPEN	NIL	NIL	NIL
17119821	MRS PRIYA SHARMA	28	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	7000	30,28	18	NORMAL	LAP	NIL	NIL	NIL
17137684	MR RAVI	31	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,28	20	NORMAL	LAP	NIL	NIL	NIL
17118778	MR VEER	68	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	8000	30,32	20	NORMAL	LAP	NIL	NIL	NIL
17152630	MR JAGMOHAN SAINI	46	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	8000	31,30	20	NORMAL	LAP	NIL	NIL	NIL
17153772	MRS RUKSANA	45	F	HCV POSITI	NORMAL	NIL	CHOLELITHIASIS	9.9	8000	45,22	20	NORMAL	LAP	NIL	NIL	NIL
17153235	MRS RAJWATI	52	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,35	20	NORMAL	LAP	NIL	NIL	NIL
17152107	MRS RAJSHREE MISHRA	25	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	13.2	6000	32,38	24	NORMAL	LAP	NIL	NIL	NIL
17158401	MR MOHAN LAL	43	M	NEGATIVE	NORMAL	PYOCELE WITH MO	CHOLELITHIASIS	10	6000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17157242	MR DEEPAK	30	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17157540	MRS RANJU	35	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17156291	MR SUNIL	27	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	7000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17158118	MRS PRIYANKA	23	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,28	28	NORMAL	LAP	NIL	NIL	NIL
17158315	MRS PREMI	48	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17152107	MRS REENA	42	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	28,27	28	NORMAL	LAP	NIL	NIL	NIL
17152167	MR MOBIN	38	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	12	6000	30,28	28	NORMAL	LAP	NIL	NIL	NIL
17159026	MRS JAYWATI	54	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	5500	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17161003	MRS SUNITA	40	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	31,30	28	NORMAL	LAP	NIL	NIL	NIL
17162501	MRS JYOTI	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	28,24	28	NORMAL	LAP	NIL	NIL	NIL
17162926	MR AKASH	23	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17162507	MRS DAYA	55	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	50,51	28	NORMAL	LAP	NIL	NIL	NIL
17163996	MRS SUNITA DEVI	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	5500	30,28	28	NORMAL	LAP	NIL	NIL	NIL
17161562	MRS SHAKIRA	30	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17169708	MRS KASHMIRI DEVI	57	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	31,30	28	NORMAL	LAP	NIL	NIL	NIL
17169510	MR NITISH	23	M	NEGATIVE	NORMAL	GB POLYP	CHRONIC CHOLECYSTI	12	7000	31,30	28	NORMAL	LAP	NIL	NIL	NIL
17157321	MRS PINKY	36	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS WITH	9	8000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17166481	MRS POOJA RAGHAV	25	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	7000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17168514	MRS VINEETA	32	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17156033	MRS CHANCHAL	28	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	5500	28,26	28	NORMAL	LAP	NIL	NIL	NIL
17169442	MRS SHAHIDA	53	F	NEGATIVE	NORMAL	TYPE 2 BILE DUCT IN	POST CHOLECYSTECTO	8.8	7000	28,30	28	NORMAL	ROUX-EN-Y	NIL	NIL	NIL
17178808	MRS REEMA	24	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	6000	30,32	28	NORMAL	LAP	NIL	NIL	NIL
17180103	MRS MAHAKASI	24	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	5000	28,26	28	NORMAL	LAP	NIL	NIL	NIL
17180019	MRS SHABNAM	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	8.8	7000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17182271	MR BALBIR SINGH	61	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	12	8000	28,26	30	NORMAL	LAP	NIL	NIL	NIL
17177535	MRS RAJWATI	62	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,35	28	NORMAL	LAP	NIL	NIL	NIL
17183439	MRS ANURADHA	22	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	8000	35,38	28	NORMAL	LAP	NIL	NIL	NIL
17185150	MRS NEERAJ	37	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	8000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17186122	MRS JAGRESH	52	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS WITH	10	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17187649	MRS PHOOLWATI	50	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	6000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17185801	MRS ASHA	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	6000	30,28	30	NORMAL	LAP	NIL	NIL	NIL
17187000	MR RAJEEV	46	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	7000	30,28	30	NORMAL	LAP	NIL	NIL	NIL
17186159	MR RAJESH	38	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17189262	MRS BABLI	50	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	9	7000	30,35	28	NORMAL	LAP	NIL	NIL	NIL
17190018	MR AJAY	42	M	NEGATIVE	ACCESSORY ARTERY IP	ACCESSORY ARTERY	CHRONIC CHOLECYSTI	12	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17190020	MRS LAKHMERI	64	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS WITH	9	7000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17192887	MRS SITARA	32	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17193877	MRS SALEEMAN	55	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,28	30	NORMAL	LAP	NIL	NIL	NIL
17192903	MRS BABITA	40	F	NEGATIVE	ACCESSORY ARTERY IP	ACCESSORY ARTERY	CHOLELITHIASIS	10	7000	30,28	30	NORMAL	LAP	NIL	NIL	NIL
17122682	MRS SAROJ	61	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	28,30	30	NORMAL	LAP	NIL	NIL	NIL
17195328	MRS SHEELA DEVI	55	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	12	7000	30,28	28	NORMAL	LAP	NIL	NIL	NIL
17194783	MR MAYANK	22	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	14	7000	30,28	28	NORMAL	LAP	NIL	NIL	NIL
17195333	MRS NEETA	32	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	7000	28,30	28	NORMAL	LAP	NIL	NIL	NIL
17194457	MS NEHA	27	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	35,38	28	NORMAL	LAP	NIL	NIL	NIL
17196139	MRS KANCHAN	41	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	7000	28,30	30	NORMAL	LAP	NIL	NIL	NIL
17196448	MRS RAJKUMARI	40	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17197344	MRS SUNDER MAVI	35	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	7000	30,35	30	NORMAL	LAP	NIL	NIL	NIL
17193676	MRS MADU	65	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	6000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17197081	MR SULTAN	45	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS WITH	14	7000	30,31	30	NORMAL	OPEN	NIL	NIL	NIL
17197523	MRS KAUSAR	42	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17198036	MRS DIMPLE	42	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	7000	30,35	30	NORMAL	LAP	NIL	NIL	NIL
17198157	MS RENU	41	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	30,35	28	NORMAL	LAP	NIL	NIL	NIL
17198683	MRS KOSAR	42	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17198187	MRS BISHAN DEVI	42	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17198157	MRS RENU	41	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	6000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17198806	MRS RATNO DEVI	60	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17198797	MRS SAVITRI	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	40,42	32	NORMAL	LAP	NIL	NIL	NIL
17198967	MR PUSHPA	40	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	8000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17199538	MRA SHIVDEVI	65	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	6000	35,30	30	NORMAL	LAP	NIL	NIL	NIL
17199268	MRS AMINA BEGUM	35	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	7000	35,30	28	NORMAL	LAP	NIL	NIL	NIL
17199907	MRS SHIVANSHI	44	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17195761	MR AYAKRAM	70	M	NEGATIVE	FROZEN CALOT	NIL	CHOLEDOCOCHOLELITI	12	9000	30,42	30	NORMAL	OPEN	NIL	NIL	NIL
17200167	MRS MEENU	26	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	11	7000	35,38	30	NORMAL	LAP	NIL	NIL	NIL
17202205	MR DAL SINGH	70	M	NEGATIVE	FROZEN CALOT	EMPYEMA	ACUTE ON CRONIC CHC	12	10000	35,38	30	NORMAL	LAP	NIL	NIL	NIL
17202405	MRS JYOTI SHARMA	28	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	40,41	30	NORMAL	LAP	NIL	NIL	NIL
17202760	MRS FATIMA	52	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	31,30	28	NORMAL	LAP	NIL	NIL	NIL
17202593	MRS MUNNI DEVI	33	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	11	6000	35,30	30	NORMAL	LAP	NIL	NIL	NIL
17202718	MRS DEEPA	28	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	35,38	28	NORMAL	LAP	NIL	NIL	NIL
17201580	MRS KAMLESH	50	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	11	8000	35,38	31	NORMAL	LAP	NIL	NIL	NIL
17202990	MR IRFAN	48	M	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	13	10000	38,39	31	NORMAL	LAP	NIL	NIL	NIL
17197312	MRS RAJANI	40	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	6000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17203415	MR FIROZ	30	M	NEGATIVE	FROZEN CALOT	EMPYEMA	CHOLELITHIASIS		10000	32,38	30	NORMAL	LAP TO OPE	NIL	NIL	NIL
17179575	MR MANOJ	40	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	6000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17203704	MR NEERAJ	32	M	NEGATIVE	FROZEN CALOT	NIL	ACUTE CALCULOUS CH	12	8000	32,38	30	NORMAL	LAP	NIL	NIL	NIL
17202803	MRS DOLLY	25	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17203851	MRS MADHURI	35	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	8000	32,38	31	NORMAL	LAP	NIL	NIL	NIL
17203937	MRS MEENA	38	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17204047	MRS SAPNA SINGH	31	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	8000	32,36	28	NORMAL	LAP	NIL	NIL	NIL
17203866	MRS MUNESH	62	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	32,36	30	NORMAL	LAP	NIL	NIL	NIL
17203826	MR MAAN SINGH	62	M	NEGATIVE	FROZEN CALOT	NIL	CHOLEDOCOCHOLELITI	12	9000	38,39	30	NORMAL	OPEN	NIL	NIL	NIL
17204038	MR JAWAHAR LAL	73	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	13	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17203858	MISS RANI	14	F	NEGATIVE	NORMAL	MICROGALLBLADD	CHOLELITHIASIS	10	6000	30,31	28	NORMAL	OPEN	NIL	NIL	NIL
17204239	MRS RADHA	38	F	NEGATIVE	FROZEN CALOT	EMPYEMA	CHOLELITHIASIS	10	10000	38,39	31	NORMAL	LAP	NIL	NIL	NIL
17204233	MRS RITA DEVI	54	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	6000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17205073	MRS KUMKUM	32	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17205265	MRS SHILPAMITI	36	F	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	10	8000	32,36	30	NORMAL	LAP	NIL	NIL	NIL
17205223	MR SHUBHAM	35	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	7000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17128279	MRS ANITA	45	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	6000	30,31	28	NORMAL	LAP	NIL	NIL	NIL
17206458	MRS GULFSHA	42	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	8000	31,30	30	NORMAL	LAP	NIL	NIL	NIL
17206175	MRS ASHA	38	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,32	30	NORMAL	LAP	NIL	NIL	NIL
17106377	MRS PUSHPA	35	F	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	10	8000	35,30	30	NORMAL	LAP	NIL	NIL	NIL
17205753	MR MEENA KHATOON	45	F	NEGATIVE	FROZEN CALOT	NIL	CHRONIC CHOLECYSTI	10	7000	38,39	30	NORMAL	LAP	NIL	NIL	NIL
17206261	MRS VINITA	22	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	7000	30,32	29	NORMAL	LAP	NIL	NIL	NIL
17209507	MRS MEENA VERMA	30	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	7000	30,31	30	NORMAL	LAP	NIL	NIL	NIL
17209634	MRS PRIYANKA	30	F	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	10	8000	30,35	28	NORMAL	LAP	NIL	NIL	NIL
17210366	MRS SAZIA	25	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	9	7000	32,36	32	NORMAL	LAP	NIL	NIL	NIL
17209902	MRS MUNNI	32	F	NEGATIVE	FROZEN CALOT	NIL	CHOLEDOCOCHOLELITI	10	8000	32,38	31	NORMAL	OPEN	NIL	NIL	NIL
17210790	MRS SUSHILA	48	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	35,38	28	NORMAL	LAP	NIL	NIL	NIL
17210524	MRS SHEELA	53	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	38,39	30	NORMAL	LAP	NIL	NIL	NIL
17212331	NR ANAND KUMAR	55	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS WITH	10	7600	32,36	31	NORMAL	LAP	NIL	NIL	NIL
17213405	MRS SONAM	32	M	NEGATIVE	NORMAL	NIL	ACUTE CALCULOUS CH	12	7900	30,32	28	NORMAL	LAP	NIL	NIL	NIL
17214184	MR VIVEK GUPTA	32	M	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	12	8000	32,36	32	NORMAL	LAP	NIL	NIL	NIL
17213921	MR KISHAN	41	M	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	12	7800	30,28	35	NORMAL	LAP	NIL	NIL	NIL
17184351	MRS RANJU	28	F	NEGATIVE	ACCESSORY ARTERY IP	NIL	CHRONIC CHOLECYSTI	10	8000	32,38	34	NORMAL	LAP	NIL	NIL	NIL
17215505	MRS JOLLY	46	F	NEGATIVE	NORMAL	EMPYEMA	CHRONIC CHOLECYSTI	10	8000	30,35	38	NORMAL	LAP	NIL	NIL	NIL
17213578	MRS EFRAZIA	52	F	NEGATIVE	NORMAL	NIL	ACUTE CALCULOUS CH	10	9000	35,38	30	NORMAL	LAP	NIL	NIL	NIL
17212980	MR YOGESH	55	M	NEGATIVE	FROZEN CALOT	NIL	CHOLELITHIASIS	12	8000	35,30	31	NORMAL	LAP	NIL	NIL	NIL
17215491	MRS BABITA	32	F	NEGATIVE	NORMAL	NIL	CHRONIC CHOLECYSTI	10	7000	35,38	35	NORMAL	LAP	NIL	NIL	NIL
17215657	MRS SAHANA	42	F	NEGATIVE	NORMAL	NECROSED GALL BL	CHOLELITHIASIS	9	8000	35,30	32	NORMAL	LAP	NIL	NIL	NIL
17216128	MR SANJAY	28	M	NEGATIVE	NORMAL	EMPYEMA	CHOLELITHIASIS	12	8000	38,39	30	NORMAL	LAP	NIL	NIL	NIL
17215530	MR KISHAN LAL	60	M	NEGATIVE	NORMAL	NIL	ACUTE CALCULOUS CH	12	8000	30,31	31	NORMAL	LAP	NIL	NIL	NIL
17217046	MRS SONIYA	40	F	NEGATIVE	NORMAL	NIL	CHOLELITHIASIS	10	8000	30,32	32	NORMAL	LAP	NIL	NIL	NIL
17217053	MRS MANJU	34	F	NEGATIVE	FROZEN CALOT	EMPYEMA	CHOLELITHIASIS	10	8000	32,36	33	NORMAL	LAP	NIL	NIL	NIL